

# REVERSE SMOKING AND PALATAL CHANGES IN FILIPINO WOMEN

Thesis submitted in partial fulfilment of the requirements for the Degree of Master of Science in Dentistry (Oral Pathology)

GEORGIANA MERCADO-ORTIZ, D.D.M.(Philippines)

DEPARTMENT OF DENTISTRY
THE UNIVERSITY OF ADELAIDE

1992

Awarded 1994

# TABLE OF CONTENTS **PAGE** iii **PRECIS** DECLARATION ACKNOWLEDGEMENTS ٧i INTRODUCTION 1 CHAPTER I REVIEW OF LITERATURE 4 CHAPTER II OBJECTIVES OF THE STUDY 51 CHAPTER III MATERIALS AND METHODS 55 CHAPTER IV RESULTS 78 CHAPTER V DISCUSSION 155 CHAPTER VI 170 CHAPTER VII CONCLUSIONS 174 APPENDICES AND REFERENCES

... to Adrianne, Cesar and Michael

#### **PRECIS**

The habit of reverse smoking is practiced in various parts of the world including the Philippines. In this preliminary cross-sectional study, 91 volunteer women smokers(61 reverse and 30 conventional) residing in nine barangays in Cabanatuan City, Philippines were interviewed and examined clinically for the presence or absence of palatal mucosal change. Seven demographic variables and twelve habit variables were investigated to characterize and compare the two study groups. The clinical examination was done to verify changes in color, texture and topography of the palatal mucosa. These changes were recorded photographically and specific features such as leukoplakic change, thickening, fissuring, pigmentation, erythema, nodularity and ulceration were observed and graded. Smears were also taken from three areas of the palate to determine cytologic features.

The majority (96.7%) of reverse smokers exhibited palatal mucosal changes including leukoplakic change, mucosal thickening, fissuring, pigmentation, nodularity, erythema and ulceration. In comparison, only 26.7% of controls exhibited mucosal changes predominantly that of intramucosal brown-black pigmentation and some erythema. This difference was statistically significant at a  $X^2$  value of 47.28 (p<.001). Analysis of the other

variables indicated that the two study groups differed significantly with regard to age(p<.05), educational attainment(p<.01), use of filtered versus non-filtered cigarettes(p<.001) and duration of smoking in years(p<.01).

The palatal changes in reverse smokers were able to be grouped into three categories. Group 1 subjects included those with pigmentation and some erythema only; Group 2 subjects(comprising the majority of reverse smokers) exhibited various combinations of mild to moderate leukoplakia, fissuring, thickening and pigmentation of the mucosa. Additional features of nodularity, erythema, prominence and reddening of minor salivary gland duct openings were also observed. Group 3 subjects included those with variable ulceration, severe reddening and non-descript roughening.

Cytologic features of smears from the two study groups did not differ as to the predominating type of epithelial cells, density and type of inflammatory cells and micronucleated cells. Only the epithelial cell type found in different areas of the palate within each group was found to be statistically significant.

#### **DECLARATION**

This thesis is submitted in partial fulfilment of the requirements for the Degree of Master of Science in Dentistry (Oral Pathology) in the University of Adelaide, Adelaide, South Australia.

This thesis contains no material which, except where due mention is made, has been accepted for the award of any other degree or diploma in any university. To the best of my knowledge, this thesis contains no material previously published published or written by another person, except where due reference has been made in the text.

Part of the material contained within this thesis has been submitted for publication to the Journal of Oral Pathology, Oral Surgery and Oral Medicine.

NAME:	GEORGIANA M. ORTIZ	COURSE:	Master of Science in Dentistre	1
-------	--------------------	---------	--------------------------------	---

I give consent to this copy of my thesis, when deposited in the University Library, being available for photocopying and loan.

SIGNATURE: DATE: 27 July 1993

Georgiana M. Ortiz

1992

To my supervisor, Dr. David Wilson, who has acted as a continous source of courage, motivation and determination, my most sincere gratitude. It has been a privilege to be among the students in oral pathology whom he has inspired and infected with the true meaning of productive research.

I am indebted to the Australian Government through the Australian International Development Assistance Bureau for the opportunity to further my studies and for the financial support extended to Filipinos like myself. It is not only the assimilation of higher education that has brought developmental change but the cross-cultural merging of ideas.

My second supervisor, Professor. Maridel P. Borja contributed important input during the planning stage with her expertise in Epideminology. Valuable technical support were given by Mrs. Sandie Powell and Mr. Peter Dent, my mentors in photography and cytology techniques. Mrs. Shirley Hastings and Ms. Mary Rhodes for taking the time out of their busy schedule to attend to my desperate word processing needs.

I would also like to extend my thanks to Professor N.C. Gervasio Dean of the College of Dentistry, University of the

Philiphines for allowing me to undertake studies in Australia and for her support and understanding.

To the Ortiz family whose moral support and patience has made this quest for higher education a process of growth. My children Adrianne, Cesar and Michael- they never asked but they understood; my father-in-law, a constant bodyguard in the field research and my husband, a constant challenge!

CHAPTER I

INTRODUCTION

#### 1.0 INTRODUCTION



Reverse smoking is a habit practiced in various parts of the world including the Indian subcontinent, Panama, Colombia, the Caribbean Islands, Venezuela, Sardinia, Sri Lanka, Jamaica, the Netherlands and some parts of Southeast Asia including the Philippines (Quigley et al., 1964; Morrow and Suarez, 1971; Pindborg, 1980; Gupta et al., 1986). This peculiar habit of placing the lighted end of the cigarette inside the mouth is practiced mostly by women in these countries (Quigley et al., 1964; Pindborg et al., 1971; Reddy et al., 1973; Bhonsle et al., 1976; Gupta et al 1984).

Women who practice the habit claim that it is a convenient method of smoking while doing housework because hot ashes are prevented from falling onto clothes while washing and on children while carrying them. It is also claimed to have a sedative effect in case of toothache because the heat generated provides a soothing sensation. Compared to conventional smoking, reverse smoking is claimed to be more economical due to an extended burning time of the cigarette. In some countries where disease carrying insects are present, this practice has also served the purpose of being an insect repellant(Quigley et al., 1964).

The origin of this habit is quite obscure and it is probably centuries old. However, it appears that mothers pass on the practice to their daughters as part of a custom. Most women who practice reverse smoking in the countries mentioned belong to the lower socio-economic group.

A wide range of benign and malignant oral mucosal changes have been associated with the habit of reverse smoking. However, while some authors claim a high malignant potential, others have observed relatively mild changes in the mouths of reverse smokers. A severe condition associated with the habit among Indian smokers is squamous cell carcinoma.

Although reverse smoking has existed in the Philippines for a long but unknown length of time, details of the habit, description of oral mucosal changes (exclusive of oral cancer) and analysis of possible risk factors related to both the habit and mucosal changes have not been investigated and described. The present community-based cross-sectional study was a preliminary survey aimed at characterization of Filipino women who practice reverse smoking and description of the range of palatal mucosal changes associated with the habit.

# CHAPTER II REVIEW OF LITERATURE

#### 2.1 TOBACCO USAGE

- 2.1.1 Origin and health implications
- 2.1.2 Forms of tobacco usage
- 2.1.3 Other risk factors associated with tobacco habits

#### 2.2 BASIC DESCRIPTION OF EPIDEMIOLOGY

- 2.2.1 Scope and relevance to dental reseach
- 2.2.2 Morbidity rates

#### 2.3 REVERSE SMOKING

- 2.3.1 Historical background
- 2.3.2 Possible aetiology of pathological changes due to reverse smoking
- 2.4 ORAL MUCOSAL CHANGES RELATED TO TOBACCO
  SMOKING HABITS (exclusive of reverse smoking)
- 2.5 ORAL MUCOSAL CHANGES RELATED TO REVERSE SMOKING

#### 2.1 TOBACCO USAGE

# 2.1.1 Origin and health implications

The late fifteenth century saw the 'introduction' of the tobacco plant to the world by the Spanish and Portuguese (Wilson, Grappin and Miguel, 1992). The term tobacco was used originally by the natives of Haiti to describe a type of a tube used by the natives for inhaling smoke from tobacco. It also refers to a cylinder of tobacco leaf prepared for smoking (IARC, 1986). The term has also been said to be derived from the Indian terms tobago or tobaca which were used to describe a Y-shaped instrument used by early American Indians for inhaling snuff (Christen et al., 1982). The term was later slightly altered by the Spaniards to "tobacco" to mean the plant and its cured leaves. Christen et al also suggested that the word "tobacco" may have had other origins, for example its possible derivation from the island of Tobago in the West Indies.

The popularity of tobacco relates to the satisfaction experienced by users of some common needs, both psychosocial and pharmacological. Psychosocial satisfaction relates to the relaxation experienced by the smoker or chewer. On the other hand, therapeutic effects ascribed to tobacco usage have included alleviation of toothache, the treatment of skin wounds and insect bites, as a tooth whitening agent and as an anti-fatigue agent when water and food are scarce (IARC, 1985; Wilson et al., 1992) However, over the centuries and particularly the twentieth increasing concern has been expressed about diseases postulated

to be associated with tobacco use. These include a wide range of medical and dental illnesses namely: respiratory diseases, cardiovascular disease, lung and other visceral cancers, leukoplakia, periodontal disease, oral and pharyngeal squamous cell carcinoma. This has caused international concern and at some periods (mostly during the sixteenth and seventeenth centuries) drastic moves by governments and religious groups against its use. For example increased taxes, imprisonment of users, starvation, beheading and other forms of physical punishment (Christen et al., 1982; IARC, 1986, Wilson et al., 1992).

The worldwide concern about the ill-effects of tobacco is a continuing issue that confronts not only medical practitioners but also dentists. The predisposition of smokers to cancer of the larynx, lungs, oral cavity and bladder has been established in numerous studies and epidemiological surveys (Wynder et al., 1976; Mahboubi, 1982; Winn et al., 1984) which have also shown that the risk of cancer increases significantly with the amount of tobacco smoke inhaled. However, interindividual genetic differences in susceptibility to smoke carcinogens have been shown in clinical trials, suggesting that hereditary constitutional factors may also be involved in smoke-associated carcinogenesis.

# 2.1.2 Forms of tobacco usage

Tobacco is generally used in one of three ways - it is smoked, chewed or inhaled. The two former methods of usage are the most common. Within each form of usage there is a

variety of different implements and techniques used to either smoke or chew tobacco. In this section methods of tobacco usage are briefly reviewed to provide a general context to the practice of reverse smoking.

Oral with combustion (tobacco smoking) - This is a widespread habit practised by people from most cultures and societies throughout the world. There is wide variability in the way that different cultures use tobacco for smoking but generally, there are two major practices - conventional and reverse smoking. In both practices, either cigars or cigarettes can be used depending on individual choices or on cultural practices. Conventional smoking, also includes the use of the pipe and its variants.

- (i) Conventional smoking the more common ways of delivering tobacco smoke internally through the oral route are collectively referred to in this study as conventional smoking. There are two sub-categories of practice in this group, a major group which includes the use of pipes, cigars and cigarettes and a minor group including use of local cigarettes such as the bidi(Indonesia) and kretek(Indonesia).
- (ii) Reverse smoking a smoking habit whereby the lighted end of the cigar or cigarette is placed and kept inside the mouth throughout most of the duration of the smoking period. The origin of this habit is still quite obscure but it has been described in several cultures mostly among

women. A complete description of the habit is presented in Section 2.1. As in conventional smoking, different forms of tobacco products are used for reverse smoking.

2.1.2.1 Pipe smoking - has been cited as the earliest form of smoking tobacco. Pipe smoking dates back at least 1000 years as evidenced by prehistoric pipes found in the Mississippi Valley, U.S.A. The practice of pipe smoking was spread by sailors from America to Europe possibly as a symbol of peace and tranquility. This ancient practice has been declining since the start of the twentieth century(IARC, 1986).

Tobaccos used for pipe smoking are usually blends of 20-25 different tobacco leaves of which the most popular are those of the burley varieties with some mixture of midrib tissues, 'casings' or 'sauces' to modify the flavour of the tobacco. On average, additives comprise about 30% of pipe tobaccos (Hoffman et al., 1963).

A variety of different pipes are found in different parts of the world. A *Hooka* is an Indian type of water pipe. Usage has been described as a widely practiced form of smoking related to social communications among groups of men in various cultures. Several varieties of water-pipe mentioned in the literature are the hooka, goza, hubble-bubble, narghile and sheesha. These types are used in some parts of the Middle East, Asia, China and Africa(IARC, 1986). Basically, the tobacco smoke in these

types is filtered through water that is kept in a special receptacle and may contain aromatic substances. (Mehta et al., 1969).

A Chilum (India)(sulpa - Nepal), is a conical clay pipe, usually about 10 cm. long. The narrow end is placed in the mouth, sometimes wrapped in a small piece of wet cloth which acts as a filter. A pebble is inserted into the bowl from above to prevent the tobacco from dropping down when the pipe is filled and lighted. (Mehta et al., 1969).

A *Hookli* is a clay pipe with a rather short stem, varying from 7 cm. to 10 cm. long and used in Gujarat, India.

2.1.2.2 Cigars - consist of fillers, binders and wrappers, all of which are made of air-cured and fermented tobaccos. They have the common feature of being wrapped either in tobacco leaf, paper or reconstituted tobacco that has been treated with tobacco extract. The aroma or flavour of a cigar is largely the result of precisely controlled treatment during fermentation. This process involves the hydrolysis and oxidative deamination of the tobacco under conditions of increased humidity and temperature(IARC, 1986). Cigars vary in form, length and diameter with weights generally ranging from 1.5 to 10 grams. Cigars in their primitive form, as used by American Indians, were described as long, thick

bundles of twisted tobacco wrapped in a dried palm or maize leaf. There has been a drastic decrease in consumption of cigars over the last 15 years.

In some parts of Asia such as India, local cigars such as cheroots or chuttas are frequently associated with the habit of 'reverse smoking' (Mehtal et al., 1969; Pindborg et al., 1971) In Indonesia, tobacco may be wrapped in corn or banana leaves to produce a local cigar.

A popular form of local cigar used in India is the chutta. Chutta is a 6 - 15 cm long country-made cigar There are two broad sometimes known as a cheroot. categories described by Pindborg et al. (1971) namely, large and small. The large one measures about 8 to 9 cm. and the small one about 5 to 6 cm. The name cheroot is derived from the Tamil "caruttu", which means roll (of tobacco) (Mehta et al., 1969; Gupta et al., 1986). chutta is a coarsely prepared cigar made of tobacco grown in Srikakulam, Andhra Pradesh, India. The tobacco used is the sun-cured and locally grown variety called the "Lanka" or "Garapa" (Reddy et al., 1971). Women in the Srikakulam and Visakhapatnam districts of India use the chutta mostly in the reverse fashion while men use it in both in the reverse and conventional manner (Pindborg et al., 1971; Mehta et al., 1977).

Other forms of local cigar include the dhumti and the calilla. A dhumti is a type of cigar made by rolling leaf

tobacco in the leaf of a jackfruit tree (Artocarpus integrifolia, L.). Occasionally the dried leaf of a banana plant(Musa paradisiaca, L.) or Hansali plant (Grewia microcos, L.) may be used. The dhumti is used for both conventional and reverse smoking in Goa, India(Bhonsle et al 1976).

A calilla is a South American home-made cigar made of tobacco leaf rolled into a long thin cigar 12 to 15 cm. in length and 5 to 10 mm. in diameter. This type of cigar is popular among reverse smokers in Colombia (Morrow and Suarez, 1971).

2.1.2.3 Cigarettes of one form or another are the most commonly used form of tobacco employed for smoking. Cigarettes are composed mainly of fine-cut tobaccos wrapped in paper or in some parts of the world a maize leaf. The composition and treatment of tobaccos used in cigarettes around the world varies widely. Cigarettes manufactured in "the west" are prepared by sophisticated technology whereas those produced in developing countries may be relatively crudely prepared and contain high tar levels. The very earliest cigarettes were made by stuffing tobacco into a hollow reed or cane tube, or by wrapping crushed tobacco leaves and shreds in a corn husk, or any other available vegetable wrapper.

As with cigarettes there are types which are peculiar to certain countries of the world. Three of

significance are the Bidi(India), the Kretek(Indonesia) and sticks(New Guinea). The Bidi is described as a local cigarette (Mehta et al., 1969) popular in India because of its low cost. A comparative chemical study of bidis with a brand of unfiltered cigarettes popular in the United States revealed that a single bidi delivers about one and a half times the carcinogenic hydrocarbons delivered by a single cigarette when smoked at the rate of two puffs per contains a relatively small amount of minute. Bidis locally grown, flaked sun-dried tobacco (.2 to .3 grams) which is hand-rolled in a rectangular piece of dried temburni leaf or other leaf deemed suitable by the user. Bidis are known to be deficient in burning agents. Hence, continuous puffing is necessary which renders a high delivery of tar dosage to the user(Gupta et al., 1984).

The Kretek is a variety of local cigarette that is widely used and manufactured in Indonesia. A feature that gives the smoke a characteristic aroma is the addition of substantial quantities of cloves (Voges, 1984; Wilson et al., 1992).

Sticks are more used in Papua, New Guinea. They are made of locally grown, shredded, sun-cured tobacco called Brus. The shredded tobacco leaf is rolled in coarse paper such as newsprint or modern cigarette paper to produce a long, large cigarette locally referred to as a stick.

without combustion - The oral use of Oral use tobacco without combustion (as well as nasal use) has been estimated to exist as long as the habit of tobacco smoking. 'Smokeless tobacco' usage involves the use of either finely powdered tobacco (as snuff) or shredded leaf form tobacco for chewing. The use of smokeless tobacco still exists worldwide and during recent years, there has been a renaissance in the use of chewing tobacco and snuff in western countries. smoking tobacco, tobacco grown for the manufacture of smokeless products is of two species (N. Tabacum and N. Rustica) within the genus Nicotiana (Solanaceae). The tobacco is often processed and treated with additives and flavouring agents. It may be taken alone or in combination with one or a variety of other ingredients (for example as a constituent of a betel quid). Oral use of 'smokeless' tobacco is said to be more predominant than nasal use. With oral use, the saliva produced may either be swallowed or expectorated depending on personal preference and/or cultural practices. Stevens (1976) refers to chewing tobacco as tobacco's body, smoke its ghost and snuff, its soul.

The habit of chewing (as compared to inhalation of snuff) tobacco is the most common form of usage of smokeless tobacco. Chewing tobaccos generally require the consumer to take a portion of the tobacco product and chew it and/or place it between the buccal mucosa and gum for varying periods of time. Christen et al. (1982) proposed that American Indians were probably the first people to smoke, chew and snuff tobacco around the early part of the 1400's. The use of chewing

tobacco by these people may have been related to a number of factors including insufficient food and a belief that its use decreased hunger, thirst and fatigue. Green tobacco leaves were chewed to quench thirst in places where water was scarce. This is evidenced by Amerigo Vespucci's(IARC, 1985) report that he found Indians on Margarita Island, a small island off the coast of Venezuela, chewing a green herb known as tobacco to quench their thirst by producing a salivary flow increase.

Indians reportedly chewed tobacco leaves to whiten their teeth (Wilson et al., 1992). Among the native Americans, chewing tobacco was also thought to have several medicinal uses including alleviation of toothache and disinfection of cuts (by spitting the tobacco juice and saliva mixture onto wound). It was also believed to relieve the effects of snake, spider and insect bites (IARC, 1985).

In Europe, tobacco was regarded as an excellent prophylactic during the plague and for those who did not like smoking, chewing was an alternative. The preference for chewing as an alternative to smoking has been attributed to its association with a rather low prevalence of ill-health and to cost factors. In contrast to smokers, chewers can keep a wad of tobacco alive for several hours and, as claimed by Christen and Glover (1981), a three-ounce (85 gms) pouch of loose-leaf chewing tobacco can last a week or more for an average chewer.

In North America, tobacco is chewed in two ways - either as a large plug called a 'chaw' or as a smaller 'quid'. The

'chaw' is placed in the buccal sulcus then it is chewed. The 'quid' is placed either in the buccal sulcus or labial sulcus and tends to be held there rather than chewed. The act of chewing both types is often accompanied by frequent spitting of tobacco extract-stained saliva.

A number of special types of chewing tobacco are used in some parts of South-east Asia, India and Africa (Wilson et al 1992). These include: Khaini (India), Pattiwala tobacco (India), Mainpuri tobacco (India), Mishri (India), Zarda (India, Arab countries, Kiwam (India), Gudakhu (India), Shammah (Saudi Arabia), Nass (Iran, Soviet Central Asia), Naswar (Afghanistan, Pakistan). Details of these habits will not be elaborated upon. However in summary, the zarda is physically chewed while khaini, mishri, shammah, nass and naswar are usually just placed in the mouth in the labial or buccal sulcus or along the Some nass users place the product under the tongue. gingiva. Naswar is usually placed in the mandibular labial sulcus area, on the floor of the mouth or the dorsum of the tongue. In India, mishri and gudakhu are primarily used as tooth cleaning agents.

Snuff is another form of smokeless tobacco. Snuff is composed of finely powdered plant material primarily consisting of tobacco. It is either used plain, or as a compound mixture of powdered tobacco and other components. Its usual form of usage is oral but it can also be used nasally. Snuff usage used to be common in the western world, but its popularity has significantly declined. In non-western countries

snuff may be used in the forms described above or inhaled through the nose.

# 2.2 Basic description of Epidemiology

# a) scope and relevance to dental research

Epidemiology is the study of the distribution and determinants of diseases and injuries in human populations. Population refers to a group of people regardless of state of health (sick or not sick). Populations can be defined either by geographic boundaries such as towns, provinces and regions or by attributes such as age, sex and socio-economic Distribution describes the occurrence of conditions as reflected by the proportion of the population affected. distinguishes Epidemiology from other sciences concerned with disease such as Pathology which is concerned with the nature of disease. Determinants are factors responsible for the observed distribution of a disease. Silverman (1985) referred to Epidemiology as that branch of science which is concerned with the study of disease as it appears in its natural surroundings and as it affects a community of Christie, Gordon and Heller (1987) formally defined it as the study of the distribution and determinants of disease in human population as it appears in its natural surroundings and as it affects a community of people.

Epidemiologic studies yield important information pertaining to:

- a) types of illnesses and their frequencies
- b) injuries in groups of people
- c) factors that influence the distribution of disease/conditions.

Their purpose is to enable us to gain a greater understanding of the natural history of disease and to develop and evaluate strategies for the prevention of disease. Epidemiologic studies also help in the ordering of priorities of resource allocation in health care and research (Christie et al., 1987). Epidemiologists work with populations in a community, not individuals and the studies involve mostly observational data. In spite of the limitations imposed by this, epidemiology is important because ultimately human disease needs to be studied in people rather than in animals and further, in people living in their community rather than lying in a hospital bed (Christie et al., 1987).

The methods of epidemiology applied to medicine are also appropriate for dental conditions and diseases. One field of endeavour that has gained considerable attention in dentistry is the identification of individuals or groups at high risk of certain diseases. This is based on findings that dental conditions, like other ailments, are not distributed randomly in the population (Beck, 1990). This implies that dental

conditions have a higher prevalence in certain groups with some common characteristics. The importance of identifying high risk groups relates to early detection, prevention and treatment of disease and the proper planning of cost-effective health programs.

Risk has been defined as the probability of an individual developing a certain condition or experiencing a health status change over a specified period of time (Kleinbaum, Kupper and Morgenstern, 1982). probability can vary from zero to one, has no dimensions and usually involves a certain reference time period. In simple terms, risk denotes the number of new cases of disease (incidence) in individuals who were exposed to the risk factor of interest over a period of time (Beck, 1990). Several rates can be calculated to indicate risk of a condition. variation is attributed to what denominator is considered or desired by a researcher. For example the person-time incidence rate. the instantaneous incidence (Morgenstern, Kleinbaum and Kupper, 1980), the force of morbidity (Chaing, 1968) and the cumulative incidence rate (Miettinen 1976; Morgenstern et al., 1980). For the purpose of this study, only two of the most commonly used rates in dentistry will be discussed briefly - the incidence and the prevalence rates.

Incidence basically refers to the number of newly developed cases at a certain point or over a period of time (Selvin, 1991). It is expressed in two ways: as incidence

rate or as incidence proportion. Incidence rate describes the number of new cases of illness over a period of time divided by the person time-at-risk. Incidence proportion is more commonly used and refers to the number of new cases of illness over a period of time divided by the number of persons at risk at the beginning of the time In the case of both the incidence rate and period. incidence proportion, an important element in calculation of the rate is time. The disease incidence rate is now usually measured in person-years, which implies accumulation of cases over a time period. For example the number of new oral cancer cases in 1991 divided by the number of person years of risk during that year will give the incidence rate for that disease. The incidence proportion is frequently a measure of risk calculated from prospective data and is unitless (Selvin, 1991). example calculation of the number of oral leukoplakic lesions in the first two years of a study divided by the number of individuals under observation will provide the incidence proportion for that condition in that population. Like all other rates, incidence proportion depends explicitly on time.

The prevalence rate is otherwise referred to as point prevalence proportion. It is the number of affected individuals in a population at a specific point in time divided by the size of the population under consideration. For example, the point prevalence proportion of oral cancer in women over 40 years of age in a specific suburb

can be computed by counting the number of existing cases divided by the number of women over 40 years of age residing in that suburb at a specified time period. difference between incidence and prevalence is that although prevalence depends on time because cases are counted at a specific time, it does not result in a value expressed per unit of time. Hence a point prevalence proportion, like all proportions is unitless. In contrast, the period prevalence proportion is the number of affected individuals in a population plus a count of new cases over a defined period of time divided by the size of the population under consideration. This measure is not commonly used since it combines both incidence and point prevalence into a single but not so meaningful rate. Selvin (1991) related incidence and prevalence by noting that when the duration of a disease is short, prevalence and incidence proportions are approximately equal; conversely for conditions with a long duration, prevalence and incidence measures most likely provide information on different aspects of a disease.

If a study aims to compare the risk of those subjects who have the potential risk factor with those who do not there are two methods which can be used to derive this information.

Table 1. Hypothetical 2 x 2 table for a potential risk factor and a dental condition.

Potential	Dental condition				
Risk factor	Yes	No	Totals		
Yes	A	В	A + B		
No	C	D	C+D		
Totals	A + C	B+D	A + B + C + D = N		

The first is the calculation of the relative risk or risk ratio (Beck, 1990). Referring to Table 1, this is calculated as (A/A+B)/(C/C+D).

The result of this computation will represent the frequency of occurrence of a disease in those who are exposed to a certain risk factor compared to those who are not. A relative risk ratio that is lesser or greater than one means that the condition is associated with the risk of disease. Incidence and relative risk are often used as indexes of health for longitudinal (follow-up) studies.

A second measure that can be used that is related to the prevalence of a disease is the odds ratio. While similar to relative risk, it also measures the association of the potential risk factor with the condition. Schlesselman (1982) described that if an event occurs with probability p, then the ratio p/q, where q=1-p, is called the odds. In the case of rare diseases, the odds and the risk of the

disease are identical, in which case the odds of having the condition in those who have the factor is A/B and for those who do not have the factor it is C/D. The odds ratio is therefore [(A/B)/(C/D)] or simply AC/BD (Beck, 1990).

The relative risk and odds ratios are primarily measures of association used in conjunction with identification and description of risk factors and high risk groups (Beck, 1990). The relative risk is usually computed from data derived from prospective studies (cohort designs), while odds ratios derive from retrospective studies such as case-control and/or cross-sectional studies.

Initially, identification of possible risk factors came from clinicians and researchers as they dealt with patients/subjects who had a disease or condition (Beck, To date, identification of risk factors for most 1990). dental diseases has come from observational studies such case-control and cross-sectional studies and some laboratory experiments. The main drawback of these studies however is the lack of control by the investigator of the possible risk factors and the inability to randomly assign the subjects into desired study and control groups. Hence, it is rather difficult to prove that the presumed risk factor actually was present prior to the development of the disease or condition as in the case of prospective and experimental studies.

The importance of distinguishing high risk groups and the identification of risk factors can be taken from two different but related points of view. clinician's perspective, risk factors are valuable clues that condition, while the in diagnosing a help may epidemiologist uses them to predict future cases of the While risk factor assessment is used clinically to disease. arrive at a proper diagnosis and management of a disease it can also serve as a guide in planning prevention and/or treatment programs. In both a clinical and epidemiologic context, the main goal is the promotion of health status.

#### 2.3 REVERSE SMOKING

# 2.3.1 Historical background



Figure 1: Filipino women practicing the habit of reverse smoking.

The existence of the habit of reverse smoking has long been recognized to exist in some parts of the world including India, Panama, Colombia, the Caribbean Islands, Venezuela, Sardinia, Sri Lanka, Jamaica, the Netherlands and some parts of Southeast Asia such as the Philippines(Quigley et al., 1964; Morrow and Suarez, 1971; Pindborg, 1980; Gupta et al., 1986; Hogewind, Greebe and van der Waal, 1987; Daftary et al., 1991, Toro and Brunicardi, 1991). This peculiar habit is characterized by placement of the lighted end of the cigar or cigarette inside the mouth for all or at least most of the smoking session (Figure 1). Cool air is drawn in through the unlighted end of the cigar or cigarette and smoke is expelled by the same route (Quigley, Cobb and Hunt, 1965). The cigar or cigarette is removed from the mouth to get rid of the ashes, although sometimes the ashes are swallowed. The manner of inhalation is usually shallow such that the smoke and tar products condense on the surface of the teeth and palate (Quigley et al., 1964).

The origin of this habit is quite obscure and Quigley et al (1964) assumed that it is probably centuries old. Several terms have been used for this habit. In the district of Andhra Pradesh, India, this custom is termed Adda Poga, in Tamil and Sardinia, it is known as Fogu a intru. In Netherlands Antilles, people speaking Papiamento refer to it as Huma pa den and among the Spanish speaking regions of South America, it is termed Fumar para adendro(Quigley et al 1964). In the Philippines, a variety of terms also correspond to the habit depending on the local dialect of smokers. Among the Ilokanos(northern people), they call it Ammal' and among the

Tagalogs (central region), it is referred to as Sumpak. In most of the areas mentioned, this custom is predominantly practised by women.

The perpetuation of this smoking habit from one generation to another possibly relates to the various reasons given by women who practice it. Among Caribbeans and South American people the following reasons were listed (Quigley et al., 1964):

- 1) Pleasurable sensations from the heat and tar products deposited in the oral cavity.
- It is a convenient method of smoking while performing household duties such as cooking or washing. By reverse smoking, the ashes are prevented from falling onto clothes or food.
- For economic reasons. It is claimed that in strong winds, the burning time of the cigarette could be extended from 2 to 18 minutes if the burning end is kept inside the mouth.
- 4) As an insect repellant in areas where disease carrying insects are present.

Interestingly, the literature describes that in 1935, a severe epidemic of yellow fever caused public health officials in Maracaibu, Venezuela to recommend reverse smoking as a means of repelling disease carrying mosquitoes. (Quigley et al., 1964).

In India, the habit is reported to occur mostly among women in the districts of Visakhapatnam and Srikakulam. The Srikakulam district is the most northern coastal district in Andhra Pradesh, while Visakhapatnam is a district on the east coast of India. (Mehta et al., 1969; Pindborg et al., 1971; Reddy et al., 1972; Mehta et al., 1977). In Srikakulam, the habit has been described as originating from females and that men copied the habit from the women.

The primary reason for the habit given by women reverse smokers in this district was to keep the habit of smoking a secret from their husbands. Because only the edge of the chutta protruded from the lips, it is indeed difficult to find out whether or not a person is reverse smoking especially if smoke is not blown out for some time. However, Pindborg and colleagues (1971) were of the opinion that only a little credit can be given to this claim of keeping the habit a secret because of the present extent of the practice among women. The second and third reasons given by women reverse smokers Srikakalum are consistent with those described for Caribbeans, Colombians and South Americans. These pertain to reverse smoking being adopted as a matter of convenience during housework and to prevent the hot ashes from falling on children and onto clothes. A fourth reason given relates to the sedative effect of reverse smoking in relation to toothache. Lastly, as a matter of tradition, females see their mothers and other females in the community practicing the habit so the habit becomes part of the normal way of life (Pindborg et al., 1971; Mehta et al., 1977).

In the Caribbean and South American peoples, Quigley et al. (1964) reported that the introduction of European culture and the increase in wealth caused by modern technology have caused segments of the population to achieve standards of living equivalent to or higher than those of the United States. However, other segments of the population have retained relatively primitive standards. Reverse smoking is a specific custom peculiar to the lower socio-economic groups. In a pilot study in the South America and the Caribbean islands, 250 patients were examined as part of a program to evaluate oral It was reported that all of the patients practiced reverse smoking and among them only two were males (Quigley et al., 1964). The type of tobacco product used by this group The cigarette was held by the teeth and the was the cigarette. lips were brought together to complete the seal (Quigley, Shklar and Cobb, 1966). The presence of even a small amount of moisture from the lips leaves a moist ring around the cigarette, which slows the burning time and prolongs the act of smoking.

In Maracaibu, reverse smoking of cigars was a pre-Colombian practice among Indians but with the advent of cigarettes, the habit became prevalent in poorer negro and mestizo women (Quigley et al., 1964). In Panama, a study of 13 women with epidermoid carcinoma of the hard palate revealed that all the women were reverse smokers of homemade cigars. Most of these women were said to live in the rural interior of the country (Review Quigley et al., 1964)

In Cartagena, Colombia where this habit was first described by the Spanish conquistadores, it was said to have been practised by aborigines; the main reason given for the habit was that it was a simple custom whereby children were taught by parents (Morrow and Suarez, 1971).

Like the Caribbeans, Colombian reverse smokers smoke in this manner as a matter of convenience during housework and to extend the burning time of the cigarette.

Among Colombian habituees, the most common form of cigar used for reverse smokers is the "calilla" (Morrow and Suarez, 1971). Two other forms of cigarette are also used - a non-filter commercial cigarette and the "puros" (type of cigar more commonly marketed in the U.S.). The cigar, calilla or cigarette is placed in the mouth with about two-thirds of its length protruding and it is held between the incisors. The tip of the tongue is placed against the lower incisors, the centre of the dorsum being relaxed downward, creating a cavity between the dorsum of the tongue and the hard palate. As a consequence, the lighted part of the cigar or cigarette is in the centre of the Smoke is blown out between the thinly parted lips or cavity. through the body of the cigar or cigarette itself. Occasionally, the smoker spits out the ashes but they do not swallow nor blow the smoke out through the nose. Some subjects admitted first smoking half of their calillas in the conventional manner and the remainder in the reverse manner (Morrow and Suarez, 1971).

# 2.3.2 Possible aetiology of pathological changes due to reverse smoking

The changes in oral structures associated with the habit of reverse smoking have been ascribed to heat, or tar products, or both (Quigley et al., 1964; Morrow and Suarez, 1971). Quigley and co-workers (1965) evaluated the contribution of heat to the production of lesions through measurements of surface and air temperatures in the mouth. Comparisons between temperatures in the cigarette burning zones for conventional and reverse smoking revealed a maximum temperature of 860°C among conventional smokers and 760°C among reverse smokers.

This observed lower maximum temperature (Quigley et al., 1965) in the burning zone of cigarettes used for reverse smoking was attributed to:

- 1) the larger moisture content of the tobacco,
- 2) the slower rate of combustion, as evidenced by the prolonged smoking time (max =18 minutes),
- 3) lack of tobacco pre-heating by the hot combustion gases during inhalation, and
- 4) the diminished intensity of puffing.

The results of palatal air and tissue temperature measurements for the two study groups revealed little change between the air and tissue temperatures at the roof of the mouth of conventional smokers. During conventional smoking,

these temperatures were said to be slightly less than body temperature (37°C). Among reverse smokers the measured air and tissue temperatures differed markedly. Both had much higher peak values than were found in conventional smoking. Air temperatures at the roof of the mouth as high as 120°C were observed in reverse smokers during these experiments and the temperature rise was attributed to radiated temperature from the glowing tip of the cigarette to the thermocouples used for measuring temperature. Tissue temperatures at the roof of the mouth were much lower than the air temperatures but reached as high as 64°C (Quigley et al., 1965).

The increased temperature of the oral mucosa among reverse smokers was attributed to radiant energy transferred from the glowing tip of the cigarette to the oral mucosa. rapid rise of so-called palatal air temperature suggests that the primary destructive mechanism in reverse smoking is a radiation burn. It is said that a cigar with an 860°C burning end temperature could produce as much as 0.8 cal/cm<sup>2</sup>sec infrared radiation. It is estimated that in reverse smokers, the total radiant input to the oral mucosa could be as high as 1 cal/cm<sup>2</sup> or more due to the combined effects of heat and longer exposure time (Quigley et al., 1965). To date, there have been no studies documenting the total heat flux necessary to produce a burn in the palatal tissue. However, on the basis of infrared absorption as an estimate of thermal damage, it was claimed that irreversible skin damage occurs at a critical temperature near 45°C. Below this level, reconstructive or anabolic

processes counter-balance the catabolic or destructive processes. The results of Quigley et al.'s (1965) work suggest that intraoral temperature peaks greater than this occur frequently for a habitual reverse smoker. Quigley and colleagues also suggested that when the mucosa acquires the leather-like surface seen in habitual reverse smokers, higher palatal temperatures may occur and tissue destruction could even be greater.

Morrow and Suarez (1971), in their study of Colombian reverse smokers, claimed that the mucosal changes which do not appear to be precancerous are induced through heat and desiccation, whereas cancerous lesions seem more likely to result from some substance released from the burning of tobacco and the collection of such substances in the floor of the mouth, base of the tongue or pharynx. This suggestion was made by Morrow and Suarez (1971) on the basis of the location of oral cancer cases in their sample. Furthermore, they suggested that something in the tobacco "juice" or "ash" had a carcinogenic effect stronger than the smoke. This is supported by the observation of Moore and Catlin (1967) that among conventional smokers, 75% of the oral cancers that developed were located in the so-called "draining area" or the floor of the mouth, hypopharynx, soft palate, fauces and base of the tongue.

Bastiaan and Reade (1980) determined the response of rat lip mucosa to two tobacco tar preparations combined with heat. The results of their experiment showed that in general, the more intense the irritation from tobacco tar and/or heat,

the more marked were the histologic changes. Alterations in the normal histology of the oral mucosa included increase in epithelial thickness which possibly renders the underlying sensitive submucosa less vulnerable to irritation. Application of substances at 60°C was said to enhance epithelial response. This led Bastiaan and Reade (1980) to believe that heat acts as an additional mucosal irritant which when combined with the application of tobacco tar results in a more accentuated response as compared to the application of tar or heat only.

# 2.4 Oral mucosal changes related to tobacco smoking habits (exclusive of reverse smoking)

Tobacco usage is practiced in various ways chiefly smoking, chewing and passive placement of tobacco products against mucosal surfaces. Each of these broad forms of tobacco usage has been associated with the induction of oral mucosal changes.

That tobacco smoking in its various forms may be associated with the induction of oral mucosal lesions is undisputed. These changes include benign, premalignant (precancerous) and malignant lesions having variable, but sometimes relatively similar, clinical manifestations.

In spite of considerable clarification of terminology over recent years, there still exists a degree of confusion as regards nomenclature of tobacco smoking associated oral mucosal changes. Terms such as leukokeratosis nicotina palati, nicotine

stomatitis, stomatitis nicotina and smoker's keratosis in the past have and in some cases continue to be used (i) interchangeably and (ii) in specific as well as non-specific clinical contexts. This has led to confusion which up until the significant studies in India of Gupta and colleagues (for example, Gupta et al., 1980 and Mehta et al., 1977) was compounded by the fact evidence (other that circumstantial) directly linking smoking habits and certain oral mucosal lesions was lacking. Thoma (1941) coined the term 'nicotine stomatitis' to describe a lesion observed in some tobacco smokers - chiefly heavy pipe and cigar smokers. Synonyms for this condition include leukokeratosis nicotina palati, stomatitis nicotina and pipe smoker's palate. The clinical features of nicotine stomatitis are well recognized and are described by features including the following:

- 1) the changes are generally confined to the hard palate
- 2) whitening and thickening of palatal mucosa. There may be additional superficial yellow or yellow/brown discolouration of the white mucosa.
- 3) the presence of prominent, red punctate spots which may become umbilicated.
- 4) the clinical changes described are expressed in varying degrees of severity ranging from mild to severe.
- 5) the condition only occurs on mucosa exposed to tobacco smoke and combustion products. For example, in an individual who wears an upper denture, the changes are not expressed on areas of the hard palate which are covered by the denture base material.

6) the condition is reversible upon cessation of smoking.

The histopathological features of nicotine stomatitis include acanthosis and hyperkeratosis (orthokeratosis, parakeratosis or both) of palatal epithelium, subepithelial chronic inflammation of variable intensity, chronic inflammation around the minor salivary orifices which may, additionally, show peripheral squamous metaplasia. There may be an associated sialadenitis involving palatal mucous glands (WHO, 1978).

The pathological nature of nicotine stomatitis has been the subject of some controversy. The World Health Organization (1978) expressed the view that this condition is not a precancerous lesion and this view was later reinforced by Gupta and colleagues (1980) on the basis of a large scale study of tobacco usage and mucosal change in Indian populations.

A contrary view has been expressed in the literature (for example Ramulu et al., 1973; Reddy et al., 1973). However, it is important to note that these studies related to the description of "nicotine stomatitis" in samples of reverse smokers (as described later). On the basis of their Indian studies, Mehta et al. (1977) and Gupta and colleagues (1980) suggested that the palatal changes seen in reverse smokers, while sometimes exhibiting some features of classic nicotine stomatitis, also exhibited additional clinical and histopathological features which indicated that nicotine

stomatitis was not an appropriate diagnosis to describe the clinical palatal changes seen in reverse smokers.

Other mucosal changes causally linked to tobacco smoking habits include leukoplakia and squamous cell carcinoma. Leukoplakia has been defined as "a white patch on the oral mucosa that can neither be scraped off nor classified as any other diagnosable disease" (WHO, 1978). A recommendation (Axell et al., 1984) that this definition be altered to "a whitish patch or plaque that cannot be characterized clinically or pathologically as any other disease and which is not associated with any physical or chemical causative agent except the use of tobacco" has subsequently been made.

The relationship between tobacco smoking habits (other than reverse smoking - dealt with in Section 2.5) and leukoplakia and, in turn, leukoplakia and oral squamous cell carcinoma, has been the subject of numerous studies. The evidence linking tobacco smoking as an aetiologic agent for leukoplakia has been exclusively reviewed by Christen, McDonald and Christen (1991) and by Daftary et al. (1992) who also reviewed evidence linking smokeless tobacco usage (for example, as a constituent of betel or as "chewing" tobacco). Data from studies in various parts of the world indicate that tobacco smoking is "a strong aetiologic factor in the development of leukoplakia" (Christen et al., 1991).

Detailed discussion of the evidence linking conventional smoking habits and oral leukoplakia is beyond the scope of this review. However, in a control group in the Indian studies of reverse chutta smoking, it was reported that similar lesions were described in about 10% of the conventional smokers (Pindborg et al., 1971). In an earlier study, Mehta and coworkers (1969) reported the occurrence of leukoplakia in 3.8% of conventional chutta smokers and in 7% of hookli pipe smokers. Chilum and hooka pipe smokers did not show appreciable lesions and was claimed to be due to lesser intensity of heat produced by the habits.

It is a common observation that variability in location of leukoplakic lesions relates to differences in oral tobacco habits. For example, smoking favours the development of leukoplakia in commissures and chewing favours the buccal mucosa or labial mucosa when khaini is kept in the lower labial groove (Mehta et al., 1969).

The appearance of leukoplakia has also been reported in users of smokeless tobacco (Daftary et al., 1992). The lesions were described as homogeneous with a wrinkled surface and were either slightly elevated or non-elevated with diffuse demarcation from the surrounding mucosa. The habit of bidi smoking combined with tobacco chewing was sometimes seen to induce leukoplakia and an associated hyperpigmentation (Daftary et al., 1992).

Leukoplakia is generally regarded as a precancerous lesion although it is also widely acknowledged that there is a wide range of reported transformation rates. For example, Lind (1987) in reviewing the subject cited transformation rates ranging from 13% to 18%. However, as pointed out by Daftary et al. (1992) many of the studies describing transformation rates are not directly comparable due to a number of factors including differences in sample selection and length of observation period. In their major study investigating tobacco habits and oral changes in India, Gupta et al. (1980) reported a transformation rate for leukoplakia of Because none of the non-tobacco users in this study developed oral carcinoma, these investigators were able to calculate the relative risk of leukoplakia among their populations as 5.6%. The study of Gupta et al. also indicated that the transformation rates for leukoplakia varied according to the tobacco habit practiced. Smoking alone associated leukoplakia in their study did not progress to oral carcinoma. Leukoplakia associated with chewing habits showed a higher transformation rate than leukoplakias associated with mixed (i.e. smoking and chewing) habits.

The existence of an association between smoking habits and oral carcinoma, regardless of the presence or absence of leukoplakia, has long been recognized. While much research has focussed upon tobacco smoking and chewing habits and their relationship to nicotine stomatitis, leukoplakia and oral carcinoma, tobacco usage in its various forms, including smoking, has also been linked to other mucosal and mucosal-

associated diseases. These diseases include submucous fibrosis, gingivitis, acute necrotizing ulcerative gingivitis and inflammatory periodontal disease. Detailed discussion of these entities is outside the scope of this reviewer. Readers are referred to Christen et al. (1991) for discussion of these conditions and tobacco smoking.

The results of epidemiologic studies over the years have suggested that as many as 90% of all cancers are related to environmental factors (Armstrong and Doll, 1975; Higginson, Doll, 1977; Winn et al., 1984). Although estimates of this percentage vary considerably, it appears obvious that most cancers are induced by personal habits associated with lifestyle as well as by other environmental factors. personal habits, alcohol usage and smoking are two practices which have gained much attention over recent years. (Elwood et al., 1984; Blot et al., 1988; Merletti et al., 1989). example, epidemiologic studies have established that alcohol and tobacco are major aetiologic agents in the development of cancer of the upper alimentary tract and that the duration and quantity of tobacco exposure significantly increase the risk of disease (Elwood et al., 1984; IARC, 1986; Blot et al 1988; Merletti et al., 1989).

As early as the nineteenth century, isolated reports were circulating that suggested cigar and pipe smoking caused cancer of the mouth. It was not until the twentieth century that firm evidence was established that lung cancer was directly related to cigarette smoking (IARC, 1986). In the late

twentieth century, in many countries, as many as one-third of all cancer deaths in men and 10% of those in women are attributed to cigarette smoking. At present, the current trend is for males to give up smoking. However, increasingly more women have been adopting the habit since the 1950's and '60's such that a corresponding increase in the proportion of women dying from a tobacco-related cancer has been observed.

Several authors have presented data describing various combinations of factors that affect the habit of smoking in relation to its role as an aetiologic factor for both benign and malignant lesions (for example, see Wynder and Stellman, 1977; Elwood et al., 1984; Blot et al., 1988; Merletti et al., Of these factors, alcohol usage has been described as acting synergistically with tobacco usage. For example, Blot et al. (1988) claimed a multiplicative effect of alcohol and tobacco instead of an additive effect. They reported that a 35-fold increase in risk was observed among those who consumed two or more packs of cigarettes and more than four alcoholic Cigarette, cigar and pipe smoking were drinks per day. separately implicated, although it was shown for the first time that risk was not as high among male lifelong filter cigarette smokers. (Blot et al., 1988).

Experimental evidence points to a greater potency of carcinogenic agents having a higher concentration and longer duration (Wynder, Mushinski and Spivak, 1977) Wynder et al. observed a greater tendency for the development of at least

one anomaly in a host when there was co-existence of increased potency exerted by a carcinogenic agent and a longer duration of exposure. For example, a .005% solution that produced a tumour late in an animal's life was less likely to produce a second tumour in that animal. In contrast, the chances of developing a second primary were greatly enhanced if an animal was exposed to .5% carcinogen solution earlier in life and had therefore been exposed for a longer period of time (Wynder et al., 1977).

In a population-based case-control study of cancer of the oral cavity and oropharynx, Merletti et al. (1989) observed a 4 - 6 fold increase in risk among subjects with medium, or high, average, lifetime tobacco consumption. increasing trend in risk was also observed among subjects with longer duration and who started smoking at a younger A younger start of smoking age was associated with age. higher Odds Ratios in both sexes (Merletti et al., 1989). smokers showed a reduction of risk when compared with current smokers. Cigarette and pipe smokers had similar risks (OR = 3.8), while cigar smokers with or without the combination of other tobacco products seemed to be at very high risk (OR = 14.6, lower 95% confidence limit = 4.7). Women in this study usually smoked only cigarettes. a higher risk was also suggested for subjects who smoked black cigarettes. No clear difference in risk was observed according to the proportion of filter cigarettes smoked. Furthermore, in male subjects, an effect of alcoholic beverages

was only obvious in subjects with an average daily consumption 120 or more grams of alcohol.

### 2.5 Oral mucosal changes related to reverse smoking

Palatal changes associated with the habit of reverse smoking- include a wide range of mucosal alterations including some of those previously mentioned. Variability of these changes is reflected not only in cultural differences and practices among populations surveyed but by the subjective preference of those who have conducted surveys in the different cultures. Most of the hospital-based and communitybased surveys describing the practice of reverse smoking which have resulted in computation of reliable morbidity and mortality rates have been carried out in India (Mehta et al., 1969; Pindborg et al., 1971; Reddy et al., 1972; Reddy et al., 1973; Bhonsle et al., 1976; Mehta et al., 1977; Gupta et al., 1980; Gupta et al., 1986). There are reports from the Caribbean Islands and South America and Colombia but the the sampling populations in these studies were not as extensive as those in India so the results did not include estimation of morbidity rates which could be applied to the population in general.

In Srikakulam, Andhra Pradesh, India, Pindborg et al. (1971) reported that 43.8% of people above 15 years of age were reverse smokers. The percentage of women smokers alone was 26.9% of the sampling population as compared to 16.9% of males (Mehta et al., 1969). A strong association of reverse smoking and palatal lesions such as palatal

leukoplakia, palatal preleukoplakia and leukokeratosis nicotina palati was reported (Mehta et al., 1969; Pindborg et al., 1971; Gupta et al., 1980).

The results of clinical and histologic examination of subjects in a 10-year follow-up survey (Gupta et al., 1980) led the investigators to conclude that among reverse chutta smokers and to some extent conventional chutta smokers, the palatal changes were rather diverse. Use of the terms leukokeratosis nicotina palati, leukoplakia and preleukoplakia was discontinued and replaced with descriptions of the various component characteristic of the lesions, namely:

- a) Palatal keratosis referred to diffuse whitening of the palatal mucosa which was either slight, moderate or intense.
- b) Excrescences 1-3 mm. large, elevated areas often with a central red dot marking the orifice of the palatal mucosa glands.
- c) Patches well-defined, elevated plaques, which could qualify for the clinical term leukoplakia.
- d) Red areas designated parts of the palatal mucosa which showed well-defined reddening without ulceration.
- e) Ulcerated areas characterized by deposits of fibrin.

g) Nonpigmented areas of the palatal mucosa which were clinically devoid of pigment.

It was noted by Mehta and colleagues (1977) that more than one of these components could be found in the palate of a reverse smoker. Patches, red areas and non-pigmented areas were recorded only if their diameter exceeded 5 mm. Increased pigmentation was often a feature among reverse smokers but less frequently, there was loss of pigmentation. However, this was recognized as a debatable observation because of the lack of information regarding the normal pattern of palatal pigmentation.

The overall incidence of palatal changes for reverse smokers in Srikakulam was 81.2 per 1,000 in males and 106.2 per 1,000 for females compared to 24.9 per 1,000 in males and 39.6 per 1,000 among females in the entire sample population. The incidence rate increased steadily with increasing age, reaching a peak in the age group 55 - 64 for males as well as females. There was a slight drop in the incidence rate in the age group 65 and above. The possibility of a cohort effect or artifact were considered as possible explanations for the latter observation.

For the individual palatal changes described earlier, the incidence of red areas with increasing age was more accentuated compared to the overall incidence rate. A tenfold difference was seen between the lowest and highest age group. The overall crude annual incidence rate of red areas was

reported to be 2.5 per 1,000 for males and 6.3 per 1,000 for females. All new palatal red areas (130) among females were found in reverse smokers and for males, 56 new palatal red areas were observed in reverse smokers compared to only 6 in conventional smokers.

The same increasing trend in frequency with increasing age was seen for patches. It was reported that for males, the overall incidence rate was 4.8 per 1,000 and 10.2 per 1,000 for females. Almost all patches were associated with the habit of reverse chutta smoking.

Of the various individual mucosal changes seen among Indian subjects in Srikakulam, the palatal changes appeared to be the most stable because of an observed low rate of regression with discontinuance of the habit. The most transient palatal components seen were the excrescences which had a greater tendency to regress with cessation of the smoking habit. It was suggested by Gupta et al. (1980) that excrescences were the initial manifestations of mucosal change in the palate of habituees. Patches appeared to regress relatively slowly after smoking cessation with the rate somewhere between that of keratosis and excrescences.

In Visakhapatnam, India the habit of reverse smoking has also been reported to be predominant among women (Reddy et al., 1973). The results of a field survey of 13,397 subjects showed that 50% of the women above 20 years of age and 25% of the men smoked chuttas in the reverse manner. In

general, 40.47% of the subjects above 20 years of age were reported to practice reverse smoking(Reddy et al.,1973). The palatal changes seen in the subjects were considered by Reddy et al. as stomatitis nicotina. Seventy to eighty per cent of the habituees were said to have this lesion and two per cent of those with stomatitis nicotina showed ulceration of the palate suggestive of malignancy. This led Reddy and co-workers to believe that stomatitis nicotina might be precancerous.

Although stomatitis nicotina was evident in both conventional and reverse smokers, a higher frequency was found among reverse smokers (both males and females). both the habituees of the total sampling population and those who were above 20 years of age, there was a two-fold difference in the proportion of subjects who exhibited Among male conventional smokers, 31% stomatitis nicotina. had palatal lesions compared to 69.49% of the reverse smokers In females, 45% of conventional smokers and 76% of reverse smokers showed stomatitis nicotina. The peak prevalence of the lesion in women occurred in the 21 to 50 age group while in men, it was in the 51 to 60 age group. Some subjects also smoked cigarettes and bidis in the conventional way. 16.82% of cigarette smokers and 20.88% of bidi smoker had stomatitis nicotina. These percentages were considerably lower than those found among reverse smokers (74.59%).

The frequency of palatal ulcers was also assessed by Reddy et al. (1973). These were more often observed in women than in men (ratio = 9:1) and more often in reverse

smokers than in conventional smokers. In the field survey of 1,933 reverse smokers (Reddy et al., 1973), it was observed that 1442 had stomatitis nicotina, 28 of which were toluidine blue positive (2%) indicating possible dysplasia and malignancy. All 28 positive subjects were women reverse smokers.

In a previous study of serial sections of biopsies from a hospital-based survey, it was shown that over one-third of the cases had epithelial atypia and about 2.4% had microinvasive carcinoma (Reddy et al., 1971; Reddy et al., 1972). In conventional cigar smokers, there was only mild epithelial atypia of the palatal mucosa while reverse smokers had moderate to severe atypias and microinvasive carcinomas.

The palatal gland ducts of reverse chutta smokers in Visakhapatnam were also studied for extent and nature of changes. Reddy and co-workers' reported on the changes in the ducts of the glands of the hard palate of 359 reverse smokers who clinically demonstrated what these authors described as stomatitis nicotina. Of these lesions, 135 were judged clinically dysplastic on the basis of the toluidine blue test. A histopathological examination of the biopsies revealed mild, moderate or severe changes around the openings of the palatal gland ducts. Mild lesions were characterized by squamous metaplasia of the ducts deep into the glands. Parakeratosis and thickening of the squamous epithelium around the opening of the ducts was also observed. The duct lining in these cases sometimes exhibited mild atypia.

clinical-histological lesions graded as moderate, a more evident squamous metaplasia of the ducts was reported, combined with parakeratosis and atypia. Additional features of moderate atypia were sometimes observed. The severe cases of "stomatitis nicotina" in this group histologically showed more marked atypia, loss of polarity, hyperchromatism, irregular keratinization and irregularity in cell size. It was observed that only older subjects exhibited duct obstruction with resultant dilation and acinar atrophy. A history of 4 to 5 decades of smoking was associated with these subjects. Microinvasive carcinoma was demonstrated in three of the 135 biopsies. Reddy and colleagues suggested that the ducts of the glands probably act as a convenient portal of entry for possible carcinogenic, pyrolytic products of tobacco.

The study described by Morrow and Suarez (1971) in general reported clinical changes in Colombian reverse smokers similar to those described in Indian reverse smoking samples (Reddy et al., 1971). An estimate of frequency of reverse smoking in the general population was not known. In their hospital based survey of 79 subjects, Morrow and Suarez reported that all subjects were of low educational and economic levels. About 67% of them did not have any education and only about 14% of the educated subjects had advanced beyond the third grade. Most of the subjects were above 40 years of age (85%) at the time of the survey and the duration of smoking ranged from 1 year to 69 years. In approximately three-fourths of the subjects, the history of smoking was at least 20 years.

Seventy-eight of the subjects exhibited grossly visible changes of the palate claimed to be consistent with stomatitis nicotina as described by Reddy et al. (1973). These changes were graded by Morrow and Suarez (1971) as either minimal. moderate or severe stomatitis nicotina similar to Reddy et al.'s (1973) classification. Minimal grade palatal changes (25 cases) included small elevations (about 2 mm. in diameter) scattered over the hard and soft palates. These elevations were firm to touch and centrally depressed with their apex having a red The palatal mucosa was generally smooth, soft and devoid of thickening except for the small elevation. Moderate grade changes (16 subjects) showed increased development of the small, round, centrally pitted elevations seen in mild grade In some cases these elevations coalesced. Broader elevations were demonstrated as a reddened or pale surface which was firm to touch in some cases and soft in others. There was gross thickening of the whole hard palate in this group of subjects. In subjects with severe changes (37 cases) gross thickening and pallor of the whole palatal mucosa accompanied by various irregularities of form was observed. In some cases the thickened mucosa also exhibited deep grooves and in others, multilobular papillary elevations were subjects showed areas typical of palatal Some leukoplakia. In subjects lacking teeth the gums took on a grayish or "ashen" tint.

Histologic analysis of the biopsies taken from the hard palate mucosa of Colombian subjects revealed a number of changes including thickening of the epithelium due to

acanthosis. Many cases showed hyperkeratosis, a exhibited dyskeratosis and parakeratosis. Usually, there was infiltration of subepithelial connective tissue the lymphocytes, plasma cells and macrophages and fibrosis involving palatal glands was sometimes seen. Occasional specimens exhibited the presence of an acute inflammatory infiltrate of subepithelial connective tissue. The peripheral portion of the ducts of the glands showed hyperplastic epithelium and tended to be dilated. The elevations seen clinically appeared to represent hyperplastic epithelium around the opening of a duct whose lumen was filled either with cellular debris or blood.

Sixteen cases of oral squamous cell carcinoma were diagnosed in their sample by Morrow and Suarez. The predominant sites of occurrence were the base of the tongue, the tonsillar fauces and adjacent pharyngeal mucosa. Two cases were located on the hard palate and two in the soft palate area.

In a study of reverse smoking in Carribean and South American subjects, Quigley and colleagues (1966) examined 250 individuals. The mucosa of all 250 subjects showed what Quigley et al. described as leukoplakia in varying degrees. Leukoplakia was most prevalent on the hard palate, but the lips, mucobuccal folds, soft palate and tongue were also involved. The colour of the palate varied from white to brown and additionally exhibited varying degrees of thickening and a leather-like appearance.

Quigley et al. (1964), in describing other oral features, reported that the buccal and lingual surfaces of the teeth showed heavy, black, tarlike deposits and that salivary flow was commonly decreased in reverse smokers. The gingival margins of teeth were sometimes eroded and the cementum often had small, carious lesions. The periodontium was either mildly or moderately involved with the marginal gingiva exhibiting some degree of inflammation and recession. The attached gingiva, however, appeared to have normal stippling. Full-mouth intraoral radiographs showed that bone loss was minimal. Ulceration and scarring were occasionally seen particularly on the lips, tongue and hard palate.

Twenty-one biopsies taken from the hard palate of selected specimens were histologically analysed by Quigley et al.,(1966). A range of histologic changes including prominent and frequent hyperkeratosis and accentuation of the stratum granulosum was observed. These investigators also reported occasional epithelial hyperplasia and minimal inflammation of the subepithelial connective tissue. None of the hyperkeratotic specimens showed frank dysplasia or dyskeratosis although some cases of extensive hyperplasia of the epithelium showed some evidence of increased nuclear chromaticity and pleomorphism.

### CHAPTER III

## OBJECTIVES OF THE STUDY

#### 3.0 OBJECTIVES OF THE STUDY

The palate has a greater tendency to be ignored during regular oral diagnostic screening procedures due to its anatomical position and relatively low disease prevalence. In some countries however, (mostly developing and tropical) high morbidity rates involving the palate for a range of neoplastic and non-neoplastic lesions have been reported in relation to the habit of reverse smoking (Mehta et al.,1969; Pindborg et al.,1980; Gupta et al., 1984, ). This unusual form of smoking habit whereby the lighted end of a cigar or cigarette is placed in the mouth has been linked to a range of benign and malignant palatal changes.

The existence of the practice of reverse smoking among Filipinos has been known for many years. However, descriptions of the oral changes associated with this habit have not, to my knowledge, been reported.

This preliminary survey aimed to :

- A. investigate the smoking behaviour of rural Filipino women in selected areas.
- B. relate the smoking habit practiced by the subjects with existing palatal mucosal changes.

Specifically, it was hoped that at the end of the study, the investigator would be able to:

- 1. determine the ratio of reverse smokers to conventional smokers in the sample.
- 2. compute the proportion of reverse smokers who exhibited palatal mucosal change and compare this with the proportion of conventional smokers showing mucosal change.
- 3. describe the subjects according to seven demographic variables, namely:
  - a. age
  - b. marital status
  - c. occupation
  - d. level of education
  - e. number of children
  - f. province of origin
  - g. monthly income
- 4. determine if the subjects differed significantly according to these demographic variables.
- 5. analyse other habit variables as they related to the study groups and the main smoking habit of the subjects.

- 6. list, categorize and describe the different palatal mucosal changes that were observed in the two groups.
- 7. establish sound baseline data for future hypothesis formulation.
- 8. assess oral exfoliative cytology smears from subjects.

# Chapter IV MATERIALS AND METHODS

#### 4.1 RESEARCH DESIGN

- 4.1.1 Type of study
- 4.1.2 The research area
- 4.1.3 Subject selection
- 4.1.4 Sample size estimation

#### 4.2 METHODS OF DATA COLLECTION

- 4.2.1 Interview schedule
- 4.2.2 Oral examination and photographs
- 4.2.3 Exfoliative cytology

#### 4.3 VARIABLES SPECIFICATION AND CODING

- 4.3.1 Dependent variables
- 4.3.2 Independent variables
- 4.3.3 Confounding variables
  - 4.3.3.1 Demographic variables
  - 4.3.3.2 Habit variables

#### 4.4. METHODS OF DATA ANALYSIS

- 4.4.1 Measure of disease frequency and exposure
- 4.4.2 Measures of association

#### 4.1 RESEARCH DESIGN

#### 4.1.1 Type of Study

This is the first community-based study on the habit of reverse smoking in the Philippines. Being a pilot study and based on the chronic nature of the "disease" to be investigated, the primary focus of this study was the observation of existing or prevalent cases of the disease in the study population. Considering the objectives, the time constraint on gathering of data and economic restrictions, a cross-sectional study design was chosen as the most appropriate type of the three basic epidemiologic observational designs. (Kleinbaum et al., 1982). The flow of the study followed that of Kleinbaum and coworkers(Appendix 1).

#### 4.1.2 The Research Area

Cabanatuan City is the main city of the Province of Nueva Ecija which is one of the largest rice producing Provinces in Central Luzon, Philippines. This city is approximately 116 kilometres north of the city of Manila (Figures 2, 3). The city is easily accesible from different points as it is traversed by road networks from neighboring towns and provinces. Thus, it has been considered as a vital transportation and trading centre for both agricultural and non-agricultural goods as well as a social and educational centre for the province of Nueva Ecija and neighboring Provinces.

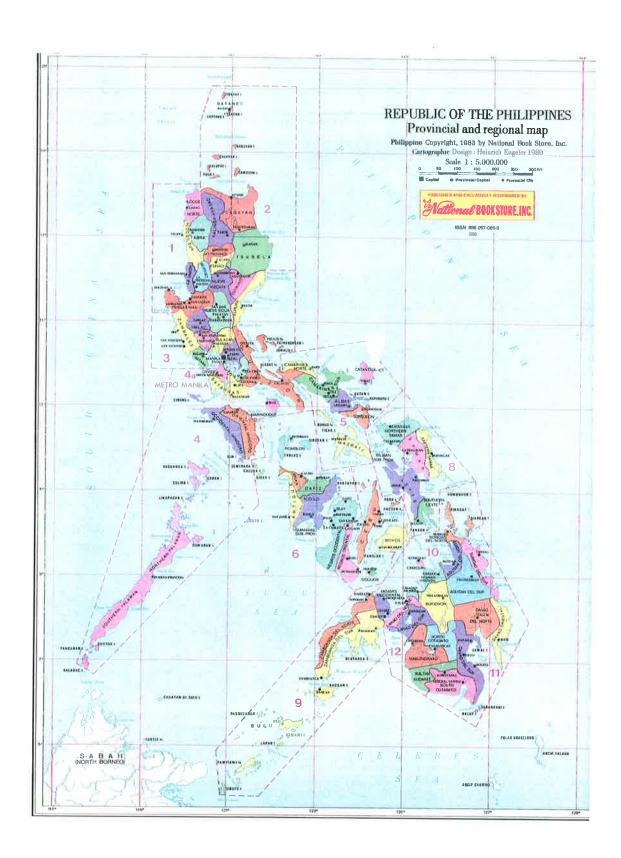
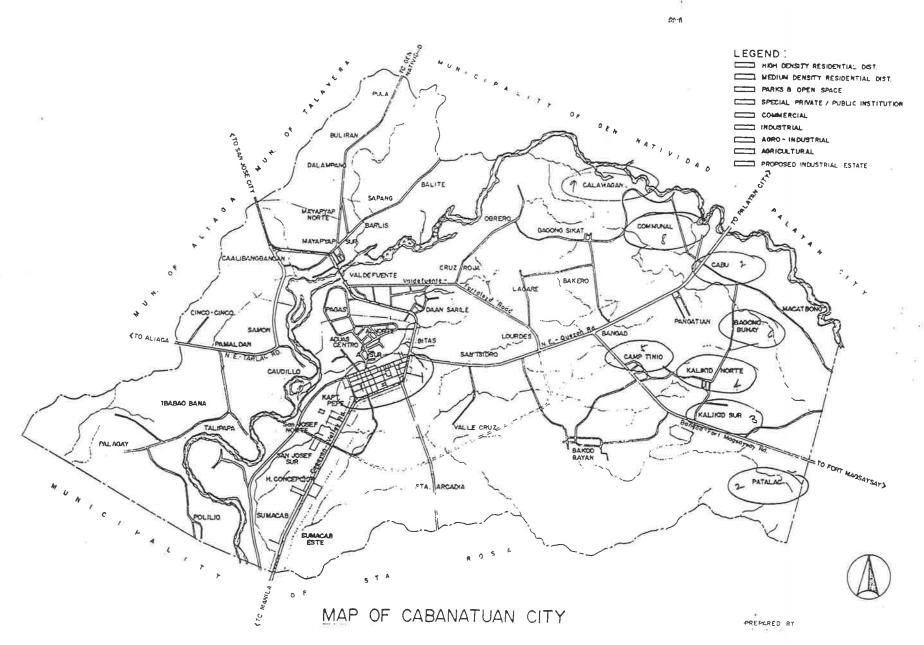


Figure 2: Map of the Philippines



The city with a land area of 19,226.63 hectares, was created on the 6th of February, 1951 by virtue of the Republic Act 526. It is considered as a second class city with a generally flat terrain which is approximately 29 meters above sea level. Like other areas of the Philippines, it has two seasons in a year - a dry season which starts in December and lasts until May, and a rainy season from June until November. The temperature varies from 15 to 38 degrees centigrade throughout the two seasons.

A total of 37 urban barangays (equivalent of suburbs) and 46 rural barangays comprise the city. As of May, 1990, the population of urban barangays totaled 86,959 for 16,781 households and that of the rural areas was recorded as 86,165 for 15,952 households. The average household size has been estimated to be approximately five.

The people of rural Cabanatuan engage in agriculture as their main source of income. In the barangays visited for this study, some residents have migrated from other places, mostly from the north. Most of the women engage in farming to augment the family income. They usually help in the planting of rice seedlings where their feet are in muddy water for the whole day during the planting season. In the heart of Cabanatuan city, trade and industry is dominated by woodcraft and handicraft, followed by metal craft, rice mills and bakeshops.

#### 4.1.3 Subject Selection

A total of nine barangays in the city of Cabanatuan, the Philippines, were chosen as research areas on the basis of advice from both local officials and health officers that the habit of reverse and conventional smoking is practiced by women in these areas. Six months prior to the actual gathering of data, pre-testing of the questionnaire was done on volunteer subjects in one of the barangays of Cabanatuan City. As a result of this pre-testing, it was learned that reverse smoking was practiced exclusively by women, mostly elderly women in the rural areas. This necessitated exclusion of males from the study in order that possible 'cases' would be matched with possible 'controls' according to sex. Such matching was done to improve the comparability of study groups (Kelsey, Thompson and Evans, 1986).

The study protocol was approved by the Human Ethics Committee of the University of Adelaide and permission was obtained from barangay chairmen (mayors) to conduct this survey prior to the work being undertaken.

Female smokers were recruited from the nine barangays on a voluntary basis. Before interview or clinical examination, a verbal explanation by the researcher regarding the background and objectives of the study was given to possible subjects in the presence of local officials and relatives or friends. Details of data collection such as the interview, oral examination, photographs and exfoliative cytology were also clarified as well

as the need for their cooperation. Subjects were assured of the confidentiality of the data. A consent form (Appendix 2) was presented to each subject emphasizing that participation in the study was voluntary and that the subject could drop-out from the study at any time.

Aside from the matching of reverse smokers to conventional smokers according to sex, inclusion of subjects took into consideration the following criteria:

- a) absence of history of oral carcinoma or other malignancies (both local and systemic);
- b) subject was either a current reverse smoker or a conventional smoker and,
- c) subject did not practice a combined smoking habit.

#### 4.1.4 Sample size estimation

Due to the absence of studies in the Philippines on precancerous lesions and smoking, and the inappropriateness of using data from research in countries such as India regarding oral cancer to predict the morbidity rate of mucosal changes among Filipino smokers, this study was conducted in two stages. The first stage involved the determination of the prevalence rate of palatal mucosa change based on 74 subjects and the determination of the sample size adequacy. Additional subjects were then added as required (refer to Appendix 3 for details of computation of sample size).

#### 4.2 METHODS OF DATA COLLECTION

Information on the dependent, independent and confounding variables were collected through interview schedule, oral examination, standard cytology methods and intraoral photographs.

#### 4.2.1 Interview schedule

The first phase of data collection was an interview of the patient by the researcher and a trained assistant using a guide questionnaire (Appendix 4) that was tested six months prior to actual data gathering. Subjects were interviewed in the presence of at least one relative, usually a spouse or a sister in order to help them recall such important events as date of birth and age when smoking was started. It would have been

preferable to validate data on date of birth by referring to the subjects' birth certificate but such documentation was not available from the majority of subjects. The interview consisted of responses to questions regarding seven demographic variables and 13 habit-related variables. In addition, the dental status and oral hygiene practices of the subjects were also assessed. The total number of smoking episodes was added to the list of dependent variables as computed by multipying duration of smoking (in years) x 365 x frequency of smoking per day.

### 4.2.2 Oral examination and photographs

The condition of the palatal mucosa of each subject was thoroughly examined under ordinary fluorescent light using 2 mouth mirrors. Changes were recorded on a proforma and with 35 mm. color transparencies. The latter was carried out using a Nikon clinical camera (Model #601M) and medical Nikor lenses (200mm, f 5.6) of 1:1 and 1:2 magnifications. transparencies were later viewed using an Evenlight Box and scored for presence of palatal mucosa leukoplakia, thickening, fissuring, nodularity, pigmentation, erythema and ulceration. For the purposes of statistical analysis, these specific features were rated using valid photographs. Valid photographs were those whose depth of field clearly showed palatal mucosa (both hard and soft) and remaining teeth where applicable. These features were scored using an ordinal rating scale (Section 4.3.1).

#### 4.2.3 Exfoliative cytology

The mucosal status of the palate of each subject was assessed on a microscopic level with the use of exfoliative cytology. An attempt was made to obtain biopsy material but strong refusals prevented the acquisition of tissue samples. For exfoliative cytology, smears were taken from three areas: anterior, middle and posterior palate. Anterior smears were taken from the rugae area, middle zone smears from the glandular area, posterior to the rugae area and anterior to the fovea palatina. Posterior smears were taken from the soft palate area.

Prior to collection of specimen, subjects were requested to rinse vigorously with plain tap water after which the areas were gently swabbed with normal saline solution. For each subject, the sampling area was scraped with a flat wooden spatula and the material transferred onto a glass slide. smears were allowed to dry slightly then fixed and stained for three hours in a solution of 2% Orcein stain in 60% Acetic acid. Subsequently smears were washed twice with absolute alcohol and then with xylene(twice) before being covered with a coverslip. The mounting medium used was Depex (Selby Anax, Subsequently they were examined using a BDH Chemicals). light microscope for a qualitative assessment of epithelial cells and inflammatory cells. Three types of epithelial cells (nucleated, non-nucleated and ghost-nucleated) (Figure 4) were distinguished and scored using an ordinal scoring system.

Inflammatory cells when present were also evaluated as to type and density.

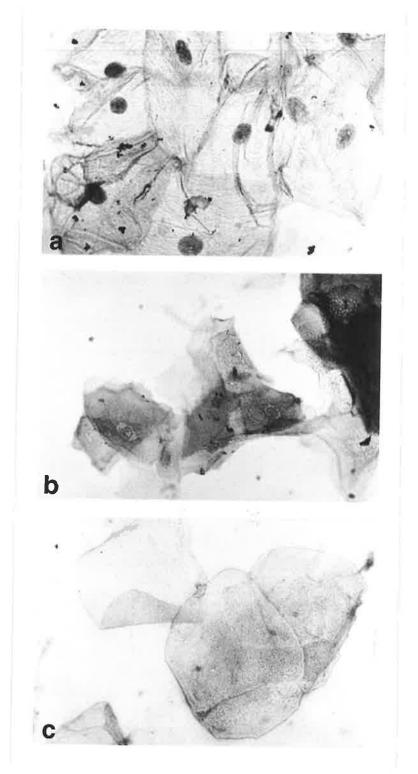


Figure 4: Photographs from smears showing (a) nucleated cells; (b) ghost nucleated cells; (c) non-nucleated cells. (Original magnification - X 60)

#### 4.3 VARIABLES SPECIFICATION AND CODING

#### 4.3.1 Dependent Variables

This group of variables refers to possible outcome variables of interest. For this study, the following macroscopic and microscopic palatal mucosal changes were examined.

- a) Macroscopic mucosal change refers to alterations in the topography, texture and colour of the palatal mucosa.

  Changes scored included the following:
  - 1) leukoplakic change
  - 2) thickening
  - 3) fissuring
  - 4) pigmentation
  - 5) nodularity
  - 6) erythema
  - 7) ulceration

The clinical features were scored using the following scale:

- 0 No change
- 1 Mild change or very slight change involving a limited area
- 2 Moderate change described involvement over approximately half to two-thirds of the palate

Severe change involving the whole palate was observed and most of the normal anatomic structures could no longer be recognized due to the gross changes.

The index of response utilized for comparison purposes was the rate of occurrence of mucosal changes among the study groups or the prevalence rate.

b) Microscopic features - involved examination of the status of surface mucosal cells as revealed by exfoliative cytology.

Six features were assessed qualitatively, namely:

- 1) the presence of micronuclei
- 2) mononuclear inflammatory cells
- 3) polymorphonuclear cells
- 4) nucleated epithelial cells
- 5) non-nucleated epithelial cells
- 6) ghost-nucleated epithelial cells

The qualitative assessment of the smears from three areas of the palate (anterior, middle and posterior) was done per subject using an ordinal measurement scale as follows:

- 0 = a cell type was not seen
- 1 a cell type was only occasionally seen

2 - numerous cells of the type being scored were observed and predominated each field encountered on a slide.(e.g. mononuclear cells versus polymorphonuclear cells).

For inflammatory cells, an overall score of '0' was given if both mononuclear cells and polymorphonuclear cells were absent. If a few of either or both cell types were seen, an overall score of '1' was given and if either or both mononuclear and polymorphonuclear cells were predominant an overall score of '2' was given.

#### 4.3.2 Independent variable

The exposure factor or determinant (Long, 1984) of interest in this research project was the type of smoking habit practised by the subjects. Two categories of smoking habit were considered:

- 1) Reverse smoking
- 2) Conventional smoking

A subject was considered a reverse smoker (also referred to as a case) if they practiced putting the lighted end of the tobacco product inside their mouth during smoking. In contrast, conventional smokers (or controls) were those who practised the usual method of having the lighted end of the cigarette outside the mouth. A code of '1' was given to reverse smokers and '0' to conventional smokers.

### 4.3.3 Confounding variables

Several other risk factors that may influence the effect of the smoking habit on the development of mucosal changes were also investigated. These risk factors are referred to in this study as possible confounding variables. Two main groups of confounding variables were considered - demographic variables and habit variables.

## 4.3.3.1 Demographic variables

Variables that gave an indication of the socio-economic status of subjects were taken as possible confounders in this study. As claimed in the literature, epidemiologists have the view that such variables can act as potential confounders because of their association with many risk factors and diseases (Liberatos Link and Kelsey, 1988). In this study the following variables were investigated:

- 1) Age of subject expressed as age in years as of most recent birthday.
- 2) Marital status subjects were categorized into one of 4 classifications: '1' for currently married subjects; '2' for single; '3' for separated and '4' for widowed.

- 3) Occupation in this study, four categories were used namely:
  - 1 farmer or farmer/housewife
  - 2 laundrywoman
  - 3 housekeeper/plain housewife
  - 4 others included business or small scale industry
- 4) An ordinal classification was used for scoring as follows:
  - 0 no schooling
  - 1 less than grade 4
  - 2 higher than grade 4 but did not finish grade 6
  - 3 finished grade 6 but not high school
  - 4 finished high school and had some college units
  - 5 finished college with or without some postgraduate education
- 5) Number of live children an indication of the economic burden of the family.
- 6) Province of origin refers to the province of origin of the subjects's immediate ancestors.

Six categories encompassed the subjects:

- 1 Ilocos region \
- Northern Philippines
- 2 Pangasinan /
- 3 Nueva Ecija \

- 4 = Bulacan } Central Philippines
- 5 = Batangas /
- 6 Visayas/Mindanao (Southern Philippines)
- 7) Income the actual gross monthly income in Philippine currency (pesos) or for farmers, the value of rice yields was computed and projected on a monthly income basis.

#### 4.3.3.2 Habit variables

- 1) Filter refers to the presence or absence of a filter in the tobacco product being used by the subject. A code of '1' was given for products with filter and '0' for those without filter.
- Source of cigarette a differentiation of manufactured cigarettes (coded as 1) versus homemade cigarettes (coded as 0) was undertaken.
- 3) Age when subject first smoked the age in years when subject had their first cigarette/tobacco puff experience.
- 4) Duration of smoking refers to the length of time that the subject had been smoking, expressed in number of years.

- 5) Frequency of smoking per day relates to the number of times the subject lit a cigarette and smoked it.
- 6) Cigarettes per day.
- 7) Inhalation practice the depth to which subject voluntarily carries smoke internally. An ordinal coding of '1' for shallow inhalation and '2' for deep inhalation was applied.
- 8) Periods of tobacco abstinence whether subjects experienced stoppage of habit within the duration of smoking years.
- 9) Months of abstinence recorded the length of time the subjects withdrew from smoking habit.
- 10) Alcohol intake a nominal variable which has been perceived to have a possible modifying effect on smoking. Subjects were coded as '1' if they took in alcohol products, '0' if not.
- 11) Alcohol with smoking indicates the combined effect of alcohol and smoking. A code of '1' was given to subjects who combined smoking and alcohol and '0' if they did not.

# 4.3.3.3 Variables of special interest

Information on reasons for preference of a certain habit and who influenced the subject to develop such practices was also gathered. Other variables such as the condition of the remaining teeth, presence of dentures where applicable and oral hygiene practice were also assessed for descriptive purposes.

#### 4.4 METHODS OF DATA ANALYSIS

#### 4.4.1 Preliminary analysis

Data generated from the interview was compiled using DBase III+ software for data management and encoding following the codelist as detailed in Appendix 5. The Microstat statistical package Version 1 was used for a preliminary statistical analysis to determine a significant difference between the two study groups based on the variables mentioned. The t-test was used for quantitative variables and either the Chi-square test or Fisher's Exact Probability test was applied where applicable to qualitative variables(Milton 1983, Daniel 1983). The collected qualitative data was cross-tabulated according to various pairs of and the resulting contingency tables were characteristics analyzed with the use of chi-square( $X^2$ ) test for association, except in those instances where the sample sizes gave rise to expected frequencies of less than 5. In those cases, the Fisher's exact probability test for 2x2 tables were used. Probabilities of at least less than 5% were considered statistically significant.

## 4.4.2 Measure of disease frequency and exposure

The point prevalence of palatal mucosal change was calculated for both groups combined and for each group under study using the following formula:

$$P_t = C_t/N_t$$

where:

P<sub>t</sub> - is the estimate of prevalence rate of mucosal change at time t.

 $C_t$  - refers to the number of existing (old and new) cases at time t.

 $N_t$  - refers to the total number of smokers. The proportion of reverse smokers (exposure rate in the sample) was calculated using the formula.

 $P_{rs} = n_{rs}/N$ 

where:

P<sub>rs</sub> - is the estimate proportion of reverse smokers among subjects interviewed.

n<sub>rs</sub> - is the sample size for reverse smokers.

N = refers to the total number of smokers eligible for statistical analysis.

#### 4.4.2 Measures of association

An assessment of exposure and disease status was carried out using the data lay-out of Kleinbaum et al. (1982) for studies involving case/non-case status as follows:

Table 2: Data lay-out for case-control and cross-sectional studies.

DISEASE STATUS	EXPOSURE CATEGORIES  No. of No. of  Exposed Unexposed		Total
No.(cases)	a	b	m 1
Non-cases	c	d	m o
Total	n 1	no	n

# a) Prevalence comparisons:

#### 1) Prevalence difference:

PDi = Pi - Po

where:

Pi - is the estimated prevalence of mucosal change among reverse smokers.

Po is the estimated prevalence among conventional smokers.

2) Prevalence ratio - this refers to the ratio of disease prevalence between exposed and unexposed groups.

# 4.4.3 Analysis of possible confounding and interaction

Variables that were found to affect the existence of palatal mucosal changes were further analyzed to verify if differences in occurence of palatal mucosal changes differed at different factor levels. A data-based analysis of confounding and interaction was carried out following the analysis detailed by Kleinbaum et al(1982) for case-control studies. This was done with the limitation in mind that results of this analysis for the present study groups were based on a cross-sectional study with a limited number of subjects which might not be representative of the population of Filipino women smokers. However, the analysis was attempted in order to have some basis for future hypothesis generation regarding suspected confounders.

The crude odds ratio  $(\widehat{cOR})$  was used to compare the effect of the "exposure" (reverse smoking) and conventional smoking on the existence of palatal mucosal changes without consideration of the different confounding variables. The hat ( $\wedge$ ) on the estimates implies that the analysis was data-based. The  $\widehat{OR}(F)$  and  $\widehat{OR}(\overline{F})$  represent the adjusted odds ratios for the

different factor levels. The presence of confounding was concluded if the crude odds  $ratio(c\widehat{OR})$  was found unequal to the adjusted odds  $ratio(a\widehat{OR})$ . Likewise, interaction was confirmed by checking for unequal estimates of effect at identified factor levels.

# CHAPTER V RESULTS

#### 5.0 RESULTS

#### 5.1 Epidemiologic features

Sixty-one(67%) of the subjects admitted smoking only in the reverse manner. The remaining 30(33%) smoked in a conventional way(Figure 5). Tables 3 - 11 give details on the frequency distribution of subjects according to the different demographic variables investigated and the corresponding results of preliminary statistical analysis. Subjects of the two study groups did not differ significantly on the basis of such variables as marital status, occupation, number of children, province of origin and monthly income (Table 12). Most of the smokers investigated were married(Table 5) with 5 to 6 children on the average(Table 9). Table 6 shows that (30)49% of reverse smokers were farmers as well as housewives while (14)46.7% of conventional smokers were housewives only.

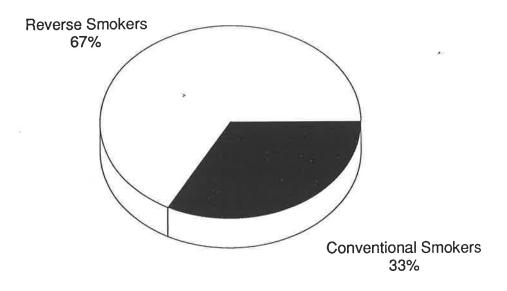


Figure 5: Distribution of subjects according smoking habit

Table 3. Distribution of subjects according to age and smoking habit

ø.	Type of smoking habit			
Age group	Conventional	Reverse	TOTAL	
15 - 34	6	0	6	
35 - 54	13	29	42	
55 and over	11	3 2	43	
TOTAL	3 0	6 1	91	

# Descriptive Statistics:

	Type of Smoking Habit		
	Conventional Revers		
Mean	50.27	56.26	
Std. Dev.	14.94	11.04	
Minimum	23	36	
Maximum	7 8	90	

Computed t value = -2.1602

p value = .0167

Table 4 Palatal changes by smoking habit by age

F	(age >39)		F	(age <40)	
_	Е	Е		Е	Е
	Reverse	Convl		Reverse	Convl
with change	57	7	with change	2	1
w/out change	1	1 6	w/out change	1	6

$$\widehat{OR}(F) = 130.3$$

$$\widehat{OR}(\overline{F}) = 12$$

Table 5 Distribution of subjects according to marital status.

	Type of smoking habit			
	Conventional	Reverse	TOTAL	
Status				
Married	29	5 5	8 4	
Single	0	1	1	
Separated	0	1	1	
Widowed	1	4	5	
TOTAL	31	61	9 1	
$X^2$ value = .457; p value = .4991				

Table 6 Distribution of subjects according to occupation and smoking habit.

	Type of smoking habit				
Occupation	Conventional Reverse TOTAL				
Farmer/Hw*	11	30	4 1		
Laundrywoman	2	5	7		
Plain housewife	1 4	2 4	3 8		
All others	3	2	5		
TOTAL	30	61	91		

 $X^2$  value = .129;

p value = .7200

\*HW - housewife

Table 7 Distribution of subjects according to highest educational level attained.

	Type of smoking habit			
Educational status	Conventional	Reverse	TOTAL	
No schooling	1	1 4	15	
< level 4	8	2 4	3 2	
>lev 4, <lev.6< td=""><td>1 2</td><td>1 8</td><td>30</td></lev.6<>	1 2	1 8	30	
>lev.6, <h.s.< td=""><td>8</td><td>5</td><td>13</td></h.s.<>	8	5	13	
>H.S.	1	0	1	
TOTAL	30	61	91	

2 X 2 Table for  $X^2$  test (education vs smoking habit):

	Conventional	Reverse	TOTAL
< level 4	9	3 8	47
> level 4	2 1	23	44
TOTAL	30	61	91

Result of  $X^2$  test:

Computed X2 value = 
$$7.155$$

p value = 
$$7.474 \text{ E}-03$$

Table 8 Palatal changes by smoking habit by educational attainment

F (ed	uc level <	or $= 4$ )	F	(educ leve	1 >4)
	Е	Е		Е	E
	Reverse			Reverse	
with change	3 7	3	with change	22	5
w/out change	1	6	w/out change	1	16
ÓR	(F) = 37		ÔR (	$(\vec{F}) = 70.4$	1

Table 9 Distribution of subjects according to number of children.

	Type of smoking habit			
<b>V</b> 2	Conventional	Reverse	TOTAL	
0 - 2	6	7	13	
3 - 5	10	1 9	29	
6 - 8	1 2	26	3 8	
9 - 11	2	8	10	
12 and over	0	1	1	
TOTAL	30	61	91	

# Descriptive Statistics

	Conventional	Reverse
Mean	5	6
Std. Dev <sup>n</sup>	3	3
Minimum	0	0
<u>Maximum</u>	9	13

Computed t value = -1.1678; p value = .1230

Table 10 Distribution of subjects according to province of origin.

	Type of smoking habit				
Province	Conventional	Reverse	TOTAL		
Northern	1 3	27	4 0		
All others	17	3 4	5 1		
TOTAL	30	61	91		
$X^2$ value = .020; p = .8881					

Table 11 Distribution of subjects according to monthly income

	Type of smoking habit			
	Conventional Reverse TOTAL			
0 - 1499	19	3 4	43	
1500 - 2999	9	2 1	3 0	
3000 - 4499	1	6	7	
over or = 4500	1	0	1	
Total	30	61	91	

Descriptive Statistics:

	Conventional	Reverse
Mean	1343.63	1190.49
Std. Dev.	2765.47	1226.59
Minimum	0	0
Maximum Computed	15,000 t value = 3668:	4,167

Table 12: Summary of preliminary statistical analysis for demographic variables

variables

results

1		
1. age	t = 2.1602;	_
2. marital status	$X^2 = .457;$	p = .4991
3. occupation	$X^2 =129$	p = .7200
4. education	$X^2 = 7.155;$	p = .0074**
5. no. of children	t = 1.168;	p = .1230
6. province of origin	$X^2 = .020;$	p = .8881
7. monthly income	t = .3668;	p = .3573

\* significant; \*\* highly significant

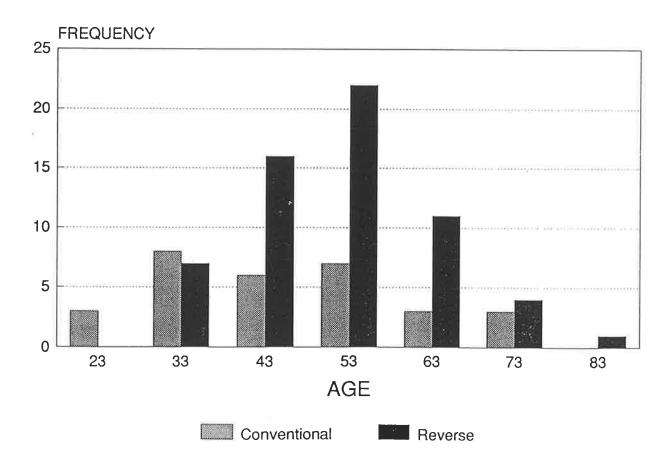


Figure 6: Distribution of subjects according to age

Of the seven demographic variables investigated, subjects of the two groups differed significantly on age and level of education attained. As shown by Table 3, reverse smokers revealed an older age range with (32)52% belonging to age group 55 and over. In contrast, (19)63% of the conventional smokers are younger than 55 years of age. Figure 6 shows the relatively even distribution of subjects for the different age groups in the controls and a contrasting greater percentage of reverse smokers for age groups 43 to 63. Results of analysis of possible confounders showed contrasting estimates of effect at various factor levels for age (Table 4). These estimates(aOR<sub>age<40</sub> = 12, aOR<sub>age>39</sub> = 130.3) are both unequal to the crude estimate(cOR=81.1)(Table 13).

Table 13 Summary table on presence or absence of palatal changes among study groups

	Type of smoking habit		
	Conventional	Reverse	TOTAL
with change	8	5 9	67
without change	2 2	2	2 4
TOTAL	30	61	91
$X^2$ value = 47.282	; pva	alue = $8.1$	92 E-11

Prev = 59/61 or 96.7%

Pcon = 8/30 or 26.7%

PR(Prevalence ratio) = .967/.267 or 3.6

cOR(crude Odds Ratio) = 81.1

Table 7 shows that (14)23% of reverse smokers had no formal education while only (1)3% of the conventionals are in the same category. Figure 7 shows the relationship of educational attainment and smoking habit. While most reverse smokers admitted attaining an education less than level 4(62%), about 70% of conventional smokers had gone beyond level 4 (Table 8). The calculated adjusted odds ratio is 70.4 for subjects who had gone beyond level 4 and 37 for those who had not gone beyond level four(Table 8). Both estimates were lower than the crude estimate which implies that an overestimation for both factor levels probably occured, i.e. the crude odds ratio indicated a stronger association between the smoking habit and the palatal changes when the level of education was not considered in the analysis.

Table 14 to 27 describe the distribution and/or crosstabulation of subjects according to habit variables. These included use of filtered versus non-filtered cigarettes, source of cigarette, age when subjects started smoking, duration of smoking in years, frequency of smoking(per day), cigarettes per day, depth of inhalation, periods of tobacco abstinence and alcohol intake. However, Table 14 shows that while (57)93.4% of reverse smokers preferred non-filtered cigarettes, only (10)33.3% of conventional smokers used them (Figure 8). The analysis for confounding showed the adjusted odds ratios of 12 for the filtered and non-filtered respectively(Table 15). Overestimation occured only for those who used filtered cigarettes but the estimation for the nonfiltered group was close to the adjusted effect. Majority of the smokers in both groups used manufactured cigarettes(Table 16).

Table 18 indicates that (32)52.4% of reverse smokers had a history of smoking from 20 to 39 years. A larger percentage of conventional smokers(13)43.3% had smoked for less than 20 years. Table 19 shows that for subjects with a duration of smoking of more than 20 years, (50/51)96% of reverse smokers exhibited palatal changes compared to (6/17)35% among conventionals. For those who had smoked for 20 years or less, 9 of 10 reverse smokers were found to have some form of palatal mucosal change while only (2 out of 13)15.4% of conventional smokers had some change. As with age, the computed adjusted risk ratios for the 2 factor levels(<21 years and >20 years) are below and above the crude estimate(Table 13) but the contrast between the adjusted estimates at the two factor levels is not as

great as that for age. A bimodal distribution of conventional smokers is shown in Figure 9 with peaks at 0 - 10 years and 30 to 40 years. For reverse smokers, a unimodal distribution is shown with the peak at 30 to 40 years.

Cross-tabulation of subjects according type of Table 14 cigarette (filtered versus non-filtered cigarette) and type of smoking habit

	Type of smoking habit		
	Conventional	Reverse	TOTAL
with filter	20	4	24
without filter	1 0	57	67
TOTAL	30	61	91

 $X^2$  value = 34.387; p value = 4.592 E-09

Table 15 Palatal changes by smoking habit by use of filtered versus nonfiltered cigarettes

F (w	ithout filte	er)	F	(with filte	er)
	E	E		Е	Е
	Reverse	$Conv^{\underline{1}}$		Reverse	Convl
with change	5 6	4	with change	3	4
w/out change	1	6	w/out change	1	16

$$\widehat{OR}(F) = 12$$

$$\widehat{OR}(\overline{F}) = 84$$

Table 16 Distribution of subjects according to source of cigarette.

	Type of smoking habit				
	Conventional Reverse TOTAL				
Home-made	2	4	6		
Manufactured	28	57	8 5		
TOTAL	30	61	91		

 $X^2$  value = .185; p value = .6675

Distribution of subjects according to age of Table 17 first puff experience.

K.	Type of smoking habit			
Age	Conventional	Reverse	TOTAL	
0 - 15	8	10	18	-
16 - 31	16	4 5	61	•
32 & above	6	6	12	
TOTAL	30	61	91	

Descriptive Statistics:

	Conventional	Reverse
Mean	2 4	21.6
Std. Dev.	13.8	7.6
Minimum	7	7
Maximum	74	42

Computed t value = .3668; p value = .357

Table 18 Distribution of subjects according to duration of smoking in years.

	Type of smoking habit  Conventional   Reverse   TOTAL			
· w/				
0 - 19	13	8	21	
20 - 39	11	3 2	43	
40 & above	6	2 1	27	
ŢOTAL	3 0	61	91	

# Descriptive Statistics:

	Conventional	Reverse
Mean	26.2	34.3
Std. Dev.	17.7	13.9
Minimum	0	4
<u>Maximum</u>	62	72

Computed t value = .3668; p value = .357

Table 19 Palatal changes by smoking habit by duration of smoking

tion > 20	years	Duration	n < 20 ye	ears
. Е	Е		E	E
Reverse	Convl		Reverse	Conv1
50	6	with change	9	2
1	1 1	w/out change	1	1 1
	Reverse	Reverse Convl 50 6	E E  Reverse Convl  50 6 with change w/out	E E E E Reverse Convl with change 9 w/out

 $\widehat{OR}(F) = 49.5$ 

 $\widehat{OR}(\overline{F}) = 91.67$ 

	Type of smoking habit		
	Conventional	Reverse	TOTAL
< 3	2	3	5
3 - 5	13	20	3 3
6 - 8	2	10	1 2
9 - 11	5	1 8	23
12 & above	8	8	16
TOTAL	3 0	61	9 1

# Descriptive Statistics:

	Conventional	Reverse
Mean	9.13	8.05
Std. Dev.	6.8	4.8
Minimum	2	2
Maximum	20	20

Computed t-value = .88; p value = .191

Table 21 Distribution of subjects according to average daily consumption in number of cigarettes.

	Type of smoking habit		
	Conventional Reverse TOT		TOTAL
1 - 5	15	27	4 2
6 - 10	7	25	3 2
11 - 15	- 1	2	3
16 & above	7	7	14
TOTAL	30	61	9 1

# Descriptive Statistics:

	Conventional	Reverse
Mean	8.97	7.8
Std. Dev.	6.9	5.3
Minimum	1	1
Maximum	20	20

Computed t value = .8558; p value = .197

Distribution of subjects according to depth of Table 22 inhalation.

<b>C</b> ?	Type of smoking habit		
	Conventional Reverse TOTAL		TOTAL
Shallow	26	53	8 9
Deep	4	8	1 2
TOTAL	30	61	91

 $X^2 \text{ value} = .090 ; \quad \text{p value} = .7637$ 

Distribution of subjects according to periods Table 23 of tobacco abstinence.

	Type of smoking habit		
	Conventional	Reverse	TOTAL
with abstinence	11	3 6	47
without abstice	19	25	4 4
TOTAL	30	61	9 1

 $X^2$  value = 3.177; p value = .0747

Table 24 Cross tabulation of subjects according to alcohol experience and type of smoking habit.

	Type of smoking habit		
	Conventional Reverse TOTAL		TOTAL
with alcohol	8	1 3	2 1
w/out alcohol	2 2	4 8	70
TOTAL	3 0	6 1	91

 $X^2$  value = .093; p value = .7601

Table 25 Cross tabulation of subjects according to presence or absence of combined alcohol and smoking habits.

Type of amalian hali			
	Type of smoking habit		
	Conventional	Reverse	TOTAL
combined	5	3	8
not combined	25	5 8	8 3
TOTAL	30	61	91

 $X^2$  value = 2.152; p value = .1424

Table 26 Distribution of subjects according to oral hygiene practice.

	Type of smoking habit		
	Conventional	Reverse	TOTAL
Toothbrush	23	4 0	63
Guava twigs	2	4	6
Toothbrush & Guava twigs	2	10	1 2
Gurgle	2	6	8
None	1	1	2
TOTAL	30	61	91

 $X^2$  value = .699; p value = .4030

Table 27 Distribution of subjects according to total number of smoking episodes.

	Smoking Habit		w-
	Conventional	Reverse	TOTAL
60 -> 13	16	29	
50600 ->	6	20	26
100060 ->	5	9	1 4
150060 ->	3	10	13
200060 ->	2	4	6
250060 & over	1	2	3
TOTAL	3 0	61	91

# Descriptive Statistics:

	Conventional	Reverse
Mean	86,908.5	100,680
Std. Dev.	77,590.8	70,812.8
Minimum	60	7,300
Maximum	284,700	328,500

computed t value = -.845; p value = .2002

Table 28: Summary of preliminary analysis of habit variables

variables	results
1. use of filter	$X^2 = 34.4;$ $p = 4.6^{-09***}$
2. type of cigarette	$X^2 = .185;$ $p = .6675$
3. age started smoking	t = 1.0467; p = .1490
4. duration (yrs)	t = 2.3875; p = .0095**
5. freq ( times per day	t = .8797; p =1907
6. number(per day)	t = .8558; p = .1972
7. inhalation practice	$X^2 = .090;$ $p = .7637$
8. periods of tobacco	$X^2 = 3.177; p = .0747$
abstinence	
9. alcohol use	$X^2 = .093$ p = .7601
10. alcohol + smoking	$X^2 = 2.152$ p = .1424
11. oral hygiene practice	$X^2 = .699$ $p = .4030$
12. Total episodes	t = .8449  p = .2002

<sup>\*\*</sup>highly significant
\*\*\*very highly significant

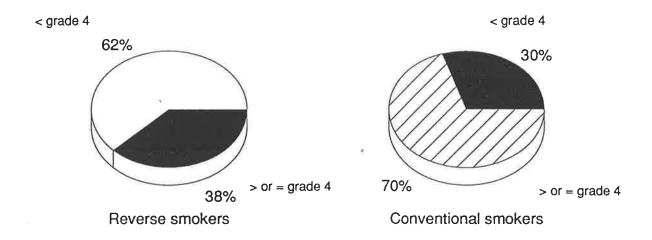


Figure 7: Distribution of subjects according to educational level attained

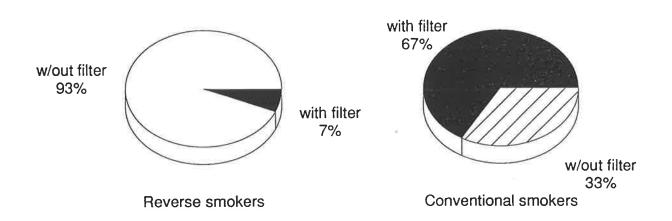


Figure 8: Distribution of subjects according to use of filtered versus non-filtered cigarettes

Table 26 shows that for the subjects with remaining teeth, the oral hygiene method used was either with the toothbrush or the guava twigs or a combination of these.

The combined effects of duration of smoking and frequency per day was also computed to give the total smoking episodes since the habit was started (Table 27). Statistical analysis did not support a suspected difference with regard to this variable.

Table 29 shows that in both study groups, subjects are mostly partially edentulous. Tables 30 and 31 give details on the reasons given by smokers for their choice of smoking habit. In Table 30, it is shown that (54)88.5% cases reportedly derived more pleasure from reverse smoking and/or reported that they could do their housework or farming uninterrupted with this method of smoking. In contrast, (19)63.3% of controls prefered conventional smoking because they were afraid of the heat produced if they reverse smoked.

Table 31 shows that (33)54% of reverse smokers were taught by their mother or other elderly women usually at the time of birth of their first child. Others acquired their habit through advise from the birth attendant(hilot)(3)5%, husband's influence(3)5%, peer influence(14)23% or spontaneously (8)13%. Thirty per cent(9) of conventional smokers were influenced by peers and only (7)23% were taught by elders

Table 29 Distribution of subjects according to status of dentition.

	Type of smoking habit				
	Conventional	Reverse	TOTAL		
Completely dentulous	3	1	4		
Partially edentulous	22	50	7 2		
Completely edentulous	5	1 0	15		
TOTAL	30	61	91		

 $X^2$  value = 3.38; p value = .1838

Table 30 Distribution of subjects according to reasons for preference of smoking habit.

	Type of smoking habit				
	Conventional Revers		TOTAL		
Greater satis- faction and/or ability to work continously	1	5 4	55		
Stronger	1	4	7		
Milder	1	0	1		
Fear of heat	19	0	19		
Non-specific	8	3	1 1		
TOTAL	30	61	91		

 $X^2$  value = 73.064; p value = 1.19 E-12

Table 31 Distribution of subjects specifying how habit was developed/acquired.

3	Type of smoking habit				
	Conventional	Reverse	TOTAL		
Taught by mother, elders	7	3 3	30		
Advised by birth attendant	2	3	5		
Influenced by husband	6	3	9		
Influenced by peers	9	1 4	23		
By ownself	6	8	1 4		
TOTAL	30	61	91		

 $X^2$  value = 6.528; p value = 4.7 E-03

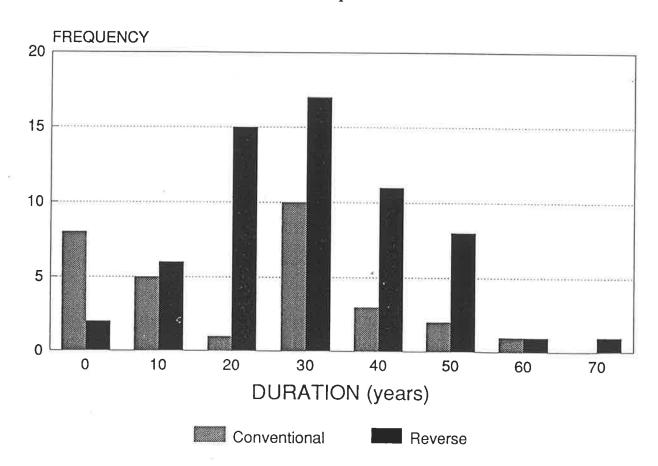


Figure 9: Distribution of subjects according to duration

## 5.2 Clinical Features

The prevalence rates of palatal mucosal change previously described (Section 5.1) were based on a dichotomous classification of changes whereby a score of "1" was given to subjects with some form of clinical change and a score of "0" was given to those with apparently normal mucosa.

Component features of palatal mucosal change were further examined and graded using valid ( see materials and methods ) 35mm. colour slides. Thirty-eight photographs from reverse smokers and thirteen from conventional smokers were viewed and grouped according to varying degrees of severity and combinations of the component changes. These changes included features of pigmentation, nodularity, thickening, fissuring, erythema, leukoplakia and ulceration. Table 32 gives the detailed distribution of photographs from the subjects according to component features present and the degree to which these features were observed based on the photographs.

The clinical changes in reverse smokers were classified into three main groups according to the predominant mucosal change observed. Group I subjects(5.3%) included those with intramucosal brown-black pigmentation and some erythema. These changes are exemplified in Figures 10 and 11. These women had a smoking history of at least 50 years and frequency of smoking was four times per day or less. Aside from the mucosal changes, poor oral hygiene was noted as evidenced by the badly carious remaining teeth.

Table 32 Distribution of subjects according to smoking habits and palatal changes.

		REVERSE			CONVENTIONAL*	
	(0)	(1)	(2)	(3)	(0)	(1)
Leukoplakia	2	11	23	2	13	0
Thickening	1	10	17	10	13	0
Fissuring	6	17	13	2	13	0
Pigmentation	22	13	13	2	7	6
Nodularity	3 0	6	2	0	13	0
Erythema	3 4	2	2	0	13	0
Ulceration	3 6	0	0	2	1 3	0

<sup>(0)</sup> = no change; (1) = mild; (2) = moderate; (3) = severe

Group II subjects comprised the majority of the reverse smokers who exhibited change (Figures 12 to 43). Of the 38 valid photographs examined, thirty-four (89.4%) were classified into this group. Main features observed included leukoplakic change (frequently stained yellow-brown), thickening and fissuring. Within this group, three subcategories were established to describe varied additional features including intramucosal brown-black pigmentation, erythema, nodularity and/or ulceration (Table 33).

<sup>\*</sup> No subjects exhibited changes beyond mild category.

Table 33 Distribution of photographs from reverse smokers (Group II) according to component changes.

	Mild n = 7 (20.6%)	Moderate n = 19 (55.9%)	Severe n = 8 (23.5)	TOTAL N = 34
pigmentation	2	9	3	1 4
nodularity	1	2	3	6
erythema	0	3	4	7
brown patch	2	10	3	15
prominent		F 2		
duct opening	1	16	3	20
leukoplakia of labial commisure	0	1	1	2
no additional feature	3	1	0	4

The subjects included in Category 2.1 (20.6%) showed mild thickening with or without leukoplakic change but did not exhibit fissuring. (Figures 12 to 16). Subjects in Figures 12 and 13 show some thickening and loss of palatal anatomical detail but no definite leukoplakic change and fissuring. A slight brown staining covers the mucosa of subject in Figure 13. The additional features of intramucosal pigmentation and/or widening of the minor salivary gland duct openings are shown by the palates of subjects in Figures 14 and 15. Both subjects were older than 70

years and had been using unfiltered cigarettes. However, while the subject in Figure 14 had reportedly smoked for 72 years, the subject in Figure 15 had been smoking for 37 years. The changes shown in Figure 14 are possibly thought to represent regression while in Figure 15, the changes appeared progressive with deeper pigmentation on the alveolar ridge areas.

Category 2.2 (55.9%) included subjects with moderate thickening, fissuring and leukoplakic change of the palatal mucosa that involved about one-half to two-thirds of the palate ( Figures 17 to 35). The palatal mucosa of the subjects in Figures 16 to 19 represent the cases that showed minimal additional features ( eg. widened salivary gland duct openings without reddening) The subjects in Figures 17 and 18 started their habit in their early 20's while the individual in Figure 19 started at 19 years of age. The same features were exhibited in subjects shown in Figures 20 and 21 but in addition, the palatal mucosa of the subjects showed some brown pigmentation. The subject in Figure 22 who was in her early 60's, like the subject in Figure 20, showed the same features of thickening, shallow fissuring and moderate leukoplakic change of the palatal mucosa. In addition to the presence of brown staining, the soft palate is seen to have intramucosal pigmentation on the left and right sides immediately posterior to the maxillary ridges.

Figure 24 is a photograph from a 54 years old reverse smoker who had been practicing the habit for 27 years. A large nodule was seen on the midpalatal area but no erythema or pigmentation.

Prominent pigmentation of the alveolar ridge and/or palate was found in 9 cases (eg. see Figures 25, 28, 30 and 33). Figure 26 shows the leukoplakic buccal mucosa of the same subject illustrated in Figure 25 and Figure 27 shows the subject's tongue exhibiting superficial brown staining. The leukoplakic palates of the subjects in this subgroup (eg. Figures 25 to 31) were commonly covered with brown stain but the gland duct openings showed minimal widening. As in the subjects of the other categories, those in category 2.2 revealed varying durations of smoking and frequency per day.

The palatal mucosa of the subjects shown in Figures 32 and 33 represent those with some erythema of the hard palate or some parts of the soft palate. One of the subjects in Category 2.2 also exhibited leukoplakia of the buccal commisure (Figure 35).

Eight subjects (23.5%) in Group II were placed in Category 2.3 because of the relatively severe changes which were observed. These included more diffuse leukoplakic change of the palatal mucosa, deeper fissuring and thickening (Figures 36, 37, 39, 41, and 43) In addition, some of the subjects exhibited more prominent and erythematous gland duct openings (Figures 39, 40, 41, 42 and 43), The subject in Figure 38 showed diffuse intramucosal brown-black pigmentation and some nodularity. A brown stain was commonly seen covering the leukoplakic palatal mucosa of the subjects.

Group III(5.3%) (Figures 44 and 45) included subjects with ulceration and/or nondescript roughening of the palatal mucosa. The palate also exhibited variable leukoplakic change on an erythematous base.

The palatal condition of conventional smokers (Figures 46 to 49) was generally within the range of normal (Figures 46 and 47). Six of the thirteen photographs from conventional smokers were assessed as exhibiting palatal change(Figures 48 and 49) chiefly intramucosal pigmentation of varying severity.

Figure 10: Reverse smoker, 62 years of age. The subject reported a history of smoking for 51 years and smoked unfiltered cigarettes 4 times a day. The anterior half of the hard palate shows intramucosal brown-black pigmentation but without evidence of thickening, fissuring or leukoplakic change. The remaining teeth are grossly carious.

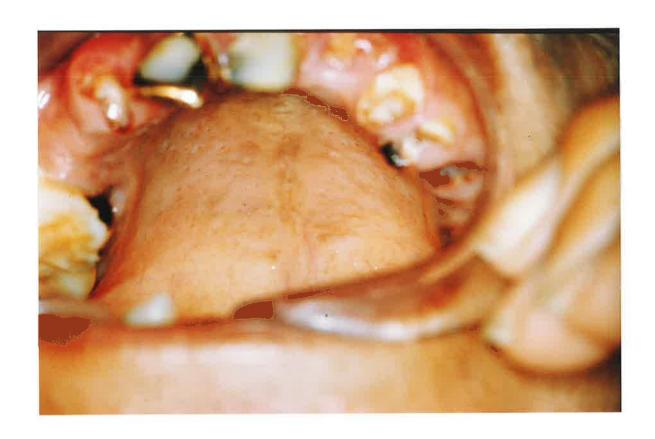
Figure 11: Reverse smoker, 90 years of age. Subject reported using unfiltered cigarettes. She started her smoking habit at the age of 29 years. She usually smoked three times per day. The mucosa of the hard palate shows intramucosal, brown-black pigmentation ( arrows ).





Figure 12: Reverse smoker, 50 years of age. Subject preferred unfiltered cigarettes and started her habit at the age of 42. She normally smokes three to four times per day. The palatal mucosa shows mild changes including slight pigmentation and widened salivary gland duct openings.

Figure 13: Reverse smoker, 63 years of age. The subject preferred unfiltered cigarettes. She started smoking at the age of 18 and smokes 10 times per day. The palate shows only mild changes of the hard palate mucosa and the rugae are appear to have retained normal morphology.



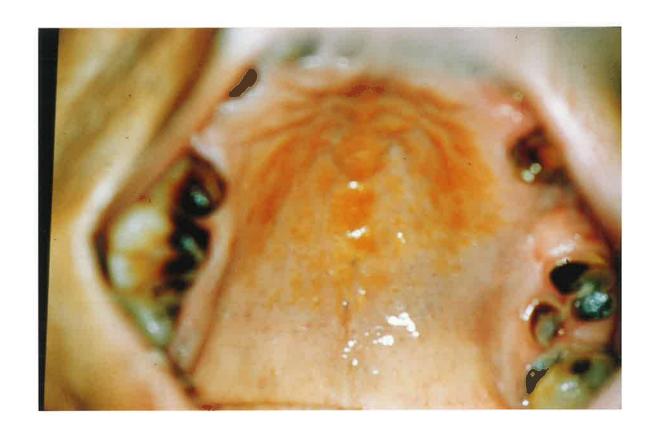


Figure 14: Reverse smoker, 80 years of age. Subject started her smoking habit at the age of eight and has been using unfiltered cigarettes since then. She used to smoke 5 to 6 times per day but at the time of the survey, she claimed to smoke only once or twice daily. The palatal mucosa shows some pigmentation of the hard palate area. It was suspected that the mucosa may have undergone regressive changes because the change in the hard palate appeared as scar-like. Leukoplakia is observed on the alveolar ridges.

Figure 15: Reverse smoker, 72 years of age. The subject had been smoking for 37 years and preferred using unfiltered cigarettes. She admitted smoking only 3 times per day. The hard palate mucosa shows intramucosal grayish pigmentation which is accentuated on the alveolar ridge areas. Some of the orifices of salivary gland ducts are slightly reddened and widened.





Figure 16: Reverse smoker, 58 years of age. The subject uses filtered cigarettes and smokes 3 time per day. She has been smoking for 46 years. The palate showed leukoplakia with some prominence and reddening of salivary gland duct opening.

Figure 17: Reverse smoker, 68 years of age. The subject preferred using unfiltered cigarettes. She started smoking at the age of 20 with a continuing frequency of 10 times per day. The whole palate shows moderate leukoplakic change with moderate thickening and mild fissuring. The palatal mucosa is unusually whitened compared to other subjects who exhibited brown staining over the thickened and whitened mucosa.



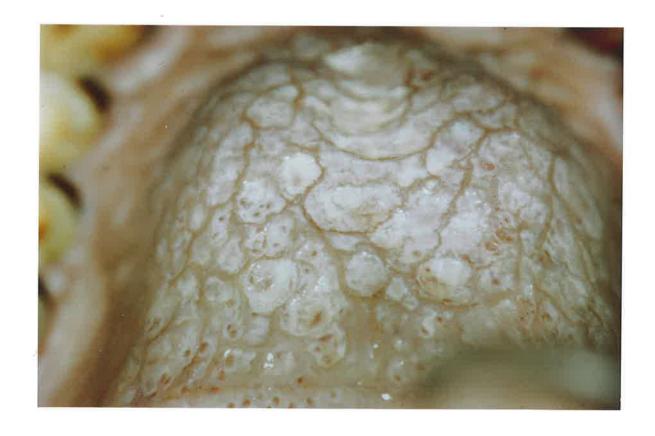


Figure 18: Reverse smoker, 50 years of age. The subject claimed to smoke 5 times per day and preferred unfiltered cigarettes. She started smoking at the age of 24. Leukoplakic change of the palatal mucosa is quite diffuse with moderate thickening fissuring. The orifices of palatal gland ducts are widened but not reddened.

Figure 19: Reverse smoker, 60 years of age. The subject preferred to smoke unfiltered cigarettes and started smoking at the age of 19 usually smoking 5 times per day. The hard and soft palate shows diffuse leukoplakic change with moderate thickening and fissuring. The lingual surfaces of the teeth exhibit heavy tar deposits.



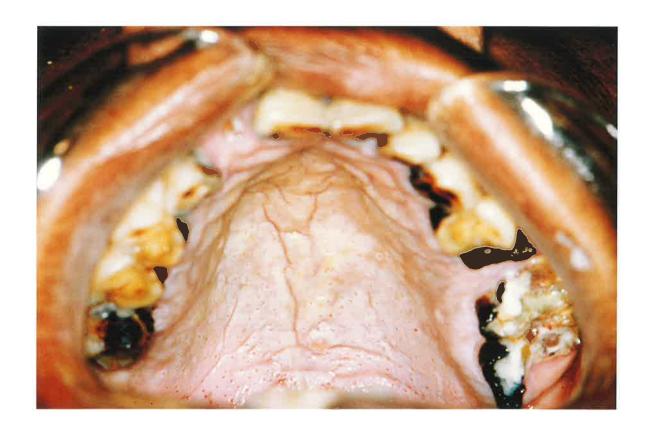


Figure 20: Reverse smoker, 64 years of age. Subject reported smoking filtered cigarettes for 43 years at a frequency of 10 times per day. The palatal mucosa shows moderate, diffuse leukoplakic change with moderate thickening and mild fissuring. The soft palate is slightly erythematous.

Figure 21 Reverse smoker, 54 years of age. The subject smokes unfiltered cigarettes. She started smoking at the age of 18 and usually smoked 7 times per day. Leukoplakic change of the palatal mucosa is moderate and diffuse with superficial brown staining extending to the soft palate and gingiva. There is moderate thickening and mild fissuring of the palate.





Figure 22: Reverse smoker, 62 years of age. Subject started smoking at the age of 24. She reported smoking 10 times per day. The palate shows severe changes including thickening, fissuring and leukoplakic change. The soft palate shows some pigmentation (arrows) and slight erythema.

Figure 23: Reverse smoker, 51 years of age. The subject reported a reverse smoking history of 17 years with a daily consumption of 5 cigarettes. The palate showed a diffuse, fissured and stained leukoplakia.





Figure 24: Reverse smoker, 54 years of age. Subject had been smoking unfiltered cigarettes for 27 years at a frequency of 10 times per day. Leukoplakic change of the palate is moderate and confined to the middle of the hard palate area. There is moderate thickening and fissuring but with relatively large nodule on the midpalatal area. As in other reverse smokers, the teeth are heavily covered with tar deposits.

Figure 25: Reverse smoker, 63 years of age. Subject began her habit at the age of 20 and has been smoking unfiltered cigarettes at an average frequency of 10 times per day. The palatal mucosa is moderately thickened with diffuse leukoplakic change extending beyond the palatal area and involving the alveolar ridge. There is also moderate fissuring and intramucosal brown-black pigmentation (arrows) on the alveolar ridge and soft palate area.



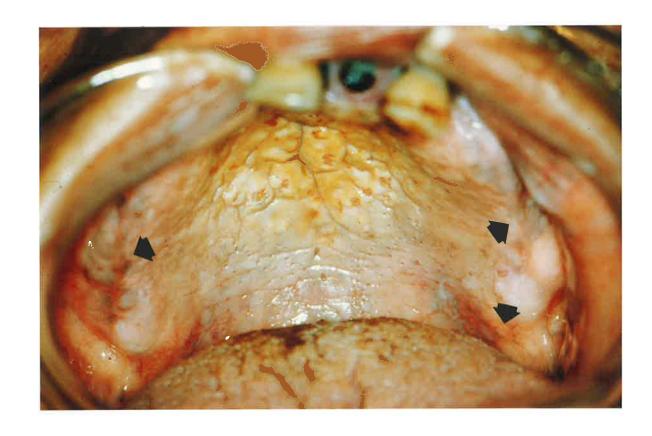


Figure 26: Same patient as in Figure 25 showing leukoplakia on the buccal mucosa of the right side corresponding to the place where the cigarette was usually held.

Figure 27: Photograph showing the tongue of the subject in Figure 25. Brown stain which could easily be scraped off is shown. Such staining was commonly found in reverse smoking subjects.





Figure 28: Reverse smoker, 44 years of age. Subject had been smoking unfiltered cigarettes since she was 18. She usually smoked 5 times per day. The palatal mucosa is moderately thickened with mild fissuring. Leukoplakic change is moderate but diffuse. Intramucosal pigmentation ( arrows ) is shown on the lingual aspect of the left alveolar ridge. The brown staining of the hard palate area, as in other subjects, could easily be scraped off.

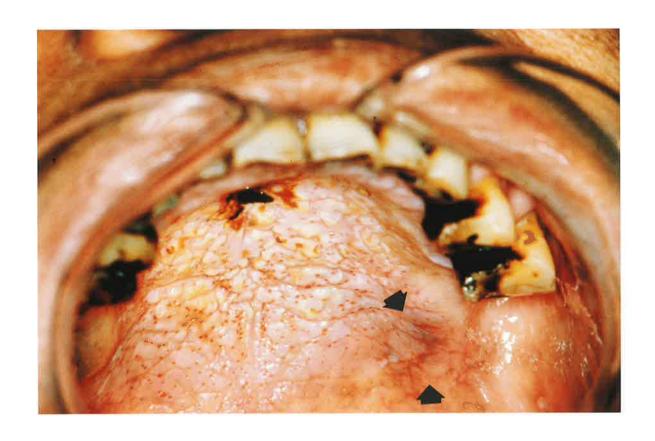
Figure 29: Reverse smoker, 60 years of age. The subject had been smoking unfiltered cigarettes for 38 years at a frequency of 2 times per day. There is moderate leukoplakic change and thickening of the palate with very mild fissuring and pigmentation. The hard palate shows some nodularity and the soft palate is mildly erythematous.





Figure 30: Reverse smoker, 60 years of age. The subject started smoking unfiltered cigarettes at the age of 16. She smokes 6 times per day. The palate shows diffuse, moderate, leukoplakic change, thickening with mild fissuring and focal pigmentation (arrows) posteriorly. There is slight erythema of the soft palate. The orifices of the palatal gland ducts are slightly widened, but not reddened.

Figure 31: Reverse smoker, 54 years of age. Subject preferred unfiltered cigarettes. She had been smoking 10 times per day since she was 19 years of age. The palatal mucosa shows moderate leukoplakic change and thickening with mild fissuring. There is slight pigmentation ( arrows ) towards the posterior area of the hard palate.



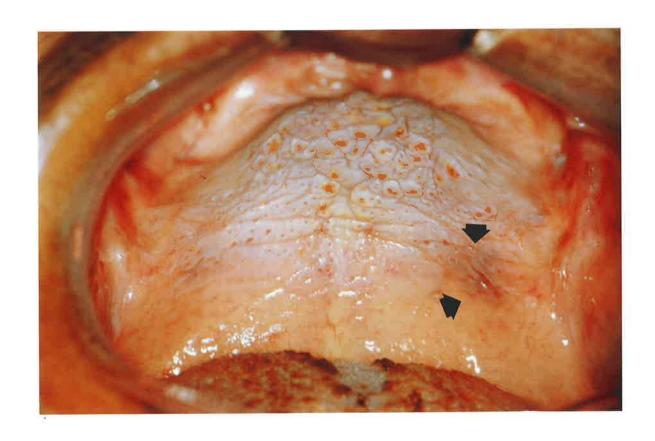
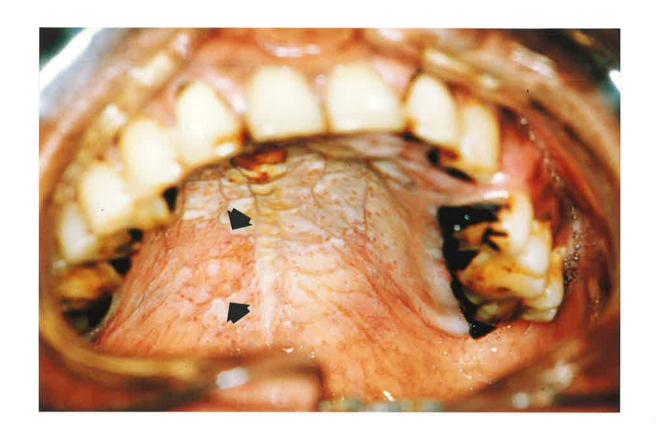


Figure 32: Reverse smoker, 56 years of age. The subject had been smoking unfiltered cigarettes for 38 years at the rate of 20 times per day. The palatal mucosa shows moderate leukoplakic change extending to the marginal gingiva, moderate thickening with mild fissuring and erythema of the soft palate. There appears to be a sloughing of part of the surface epithelium ( arrows ) on the right side of the hard palate. Lingual surfaces of the teeth show heavy tar deposits.

Figure 33: Reverse smoker, 57 years of age. Subject had been smoking unfiltered cigarettes for 34 years at the rate of 5 times per day. Leukoplakic change of the palatal mucosa is moderate but diffuse with moderate thickening and fissuring. Pigmentation (arrows) and erythema are features seen in the soft palate area.



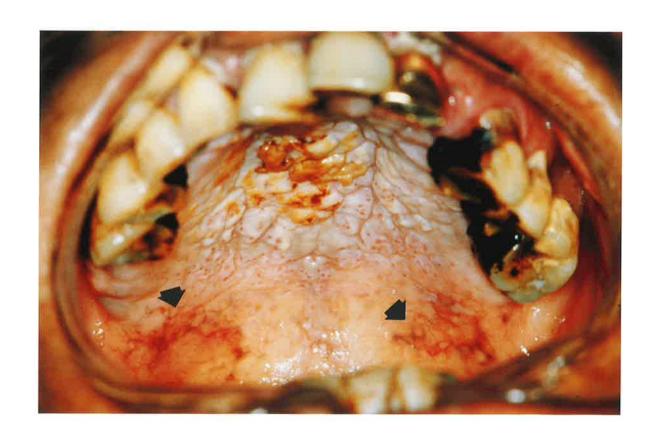


Figure 34: Reverse smoker, 55 years of age. The subject has been smoking for 45 years and smoked 20 cigarettes per day. The palate showed a diffuse, fissured leukoplakia with prominent gland duct opening.

Figure 35: Same subject as shown in Figure 21. Leukoplakia of the labial commisure is evident in the area where the subject claimed the cigarette is usually placed.





Figure 36: Reverse smoker, 71 years of age. The subject started her habit at the age of 18 and has been smoking unfiltered cigarettes at a rate of 10 to 12 times per day. Severe thickening and fissuring of the palatal mucosa is seen involving the whole palate. Leukoplakic change is diffuse. The teeth show heavy tar deposits.

Figure 37: Reverse smoker, 54 years of age who smoked unfiltered cigarette. She started her smoking habit at the age of 42 and she smokes 4 times per day. The palatal mucosa is severely thickened with moderate leukoplakic change and fissuring. The remaining teeth are encased in heavy tar deposits



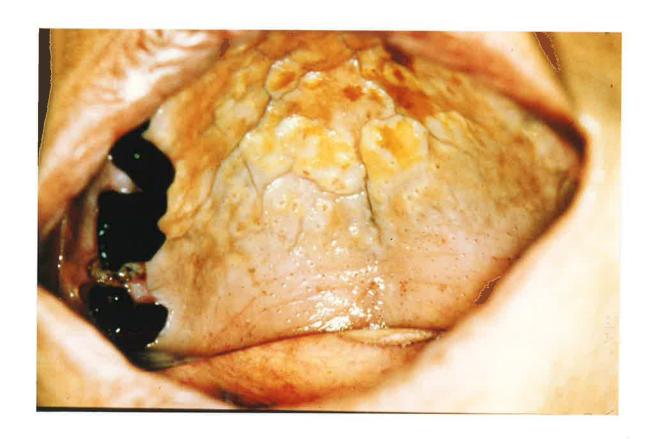


Figure 38: Reverse smoker, 45 years of age. The subject smoked unfiltered cigarettes. She started her habit at the age of 29 and smokes 8 to 10 times per day. The palatal mucosa shows diffuse intramucosal grayish pigmentation with a few of the orifices of gland ducts having accentuated thickening.

Figure 39: Reverse smoker, 56 years of age. She started smoking in the reverse manner at age 32, after giving birth to her third baby. She smokes unfiltered cigarettes at a frequency of 7 times per day. The palatal mucosa shows severe thickening and fissuring. Leukoplakic change is quite diffuse and shows brown staining. The lingual surfaces of remaining teeth show heavy tar deposits.





Figure 40: Reverse smoker, 53 years of age. Subject smokes unfiltered cigarettes and started the habit at 20 years of age. She usually smokes 10 times per day. There is severe thickening and leukoplakic change of the palatal mucosa with mild fissuring and intense intramucosal brown-black pigmentation (arrows) on the soft palate area. Small nodules are seen on the hard palate area.

Figure 41: Reverse smoker, 53 years of age. The subject prefers smoking unfiltered cigarettes. The palatal mucosa shows severe thickening, leukoplakic change and fissuring. The soft palate shows erythema and widening of glandular duct openings.

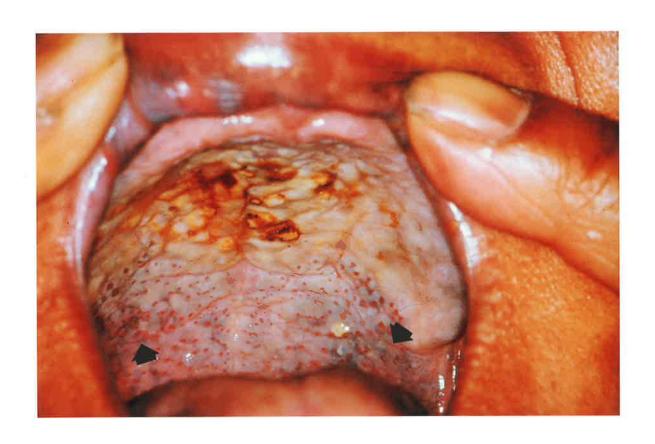




Figure 42 Reverse smoker, 50 years of age. The subject started smoking at the age of 18 and prefers non-filtered, manufactured cigarettes. She smokes every two hours on the average. Photograph shows a high magnification of the soft palate to focus on reddened, widened glandular duct orifices.

Figure 43a:Reverse smoker, 77 years of age. The subject reported smoking unfiltered cigarettes and has been smoking 8 to 10 times per day since she was 23. There is gross, severe thickening and fissuring of the palatal mucosa. Leukoplakic change of the mucosa is diffuse and extends up to the lingual half of the alveolar mucosa. The soft palate is erythematous and the orifices of the gland ducts are widened.



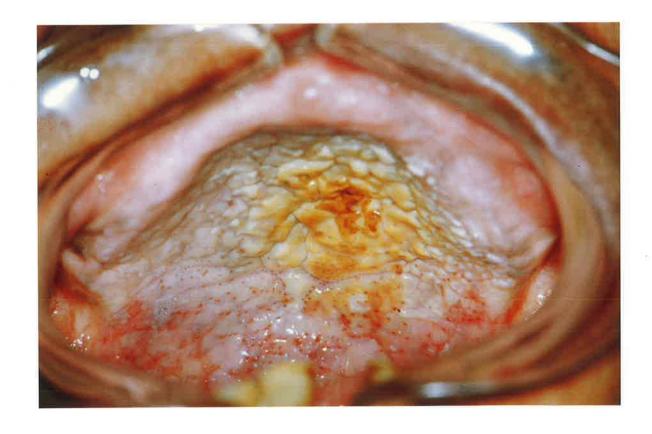


Figure 43b:Same subject featured in Figure 42 showing leukoplakia at the site corresponding to the usual placement of the end of cigarette.

Figure 44: Reverse smoker, 74 years of age. The subject prefers smoking unfiltered cigarettes. She started smoking at the age of 18 and claimed to smoke 10 times per day. The mucosa of both hard and soft palates shows severe ulceration and moderate leukoplakia on an erythematous base.

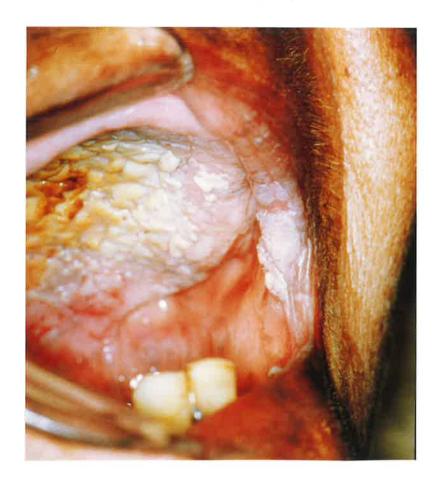




Figure 45: Reverse smoker, 48 years of age. Subject started smoking at the age of 19 and prefers unfiltered cigarettes. She smokes 20 times per day. The hard palate mucosa shows nondescript roughening and leukoplakia on an erythematous base. Some pigmentation is observed in the soft palate. The teeth are covered with tar deposits.

Figure 46: Conventional smoker, 67 years of age. The subject preferred using filtered cigarettes and started her habit at the age of 17. The palatal mucosa shows normal texture, colour and topography.





Figure 47: Conventional smoker, 54 years of age. The subject prefers filtered cigarettes and has been smoking since the age of 15 years. She smokes 20 times per day. The clinical features of the palate appear normal

Figure 48 Conventional smoker, 40 years of age. The subject uses filtered cigarettes and has been smoking for 8 years. She smokes 10 times per day. The photograph shows slight pigmentation in the midpalatal area of the mucosa (arrows).



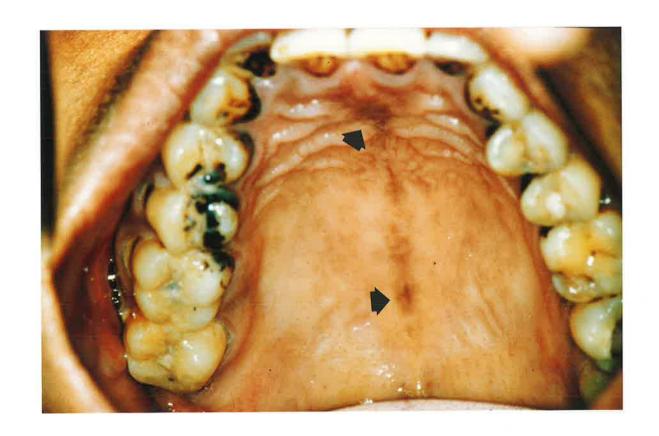




Figure 49: Conventional smoker, 33 years of age. The subject preferred using filtered cigarettes and consumed about one pack per day. She started smoking at the age of 18 years. Intramucosal grayish pigmentation was observed on the center of the hard palate.

# 5.3 Exfoliative cytology

One-hundred and thirty eight valid slides from subjects clinically graded using photographs were viewed under the light microscope for a qualitative evaluation of epithelial cells and inflammatory cells and micronuclei. As shown in Table 34, nucleated epithelial cells predominated in smears from the posterior palate areas of conventional smokers and the nonnucleated cells predominated in anterior and middle areas. Among reverse smokers, nucleated cells were more abundant in smears from the anterior and posterior areas while in mid-palate zone, non-nucleated and ghost nuclei cells were predominantly found (Table 35). This difference in predominant type of epithelial cell seen within each group was statistically significant  $[X^2 = 11.159, p<.01(conventional); X^2 = 22.503, p<.01(reverse)].$ Between the two smoking groups, the difference was not substantial enough to distinguish subjects of one group from another.

The density of micronuclei as described by Stich and Rosin (1983) in smears of the three palatal areas are detailed in Tables 36 to 38. The smears from both study groups showed similar amounts of micronuclei and although the reverse smokers revealed more in the anterior and posterior areas, the difference was not significant enough to indicate that micronuclei have the tendency to increase in reverse smokers.

Smears from conventional smokers showed no inflammatory cells in the anterior palatal area and mild to moderate infiltrate in 11 slides(21.5%) (Table 39) from of middle and 11 slides(22%) from posterior areas (Table 40). Among those who practiced reverse smoking, their smears showed mild inflammatory cell infiltrate in the anterior and middle palate areas(13 slides or 25.5%) Table 39 while the posterior area had mild to moderate infiltrate (24 slides or 48 %) (Table 40). However, the results of the Fisher's exact probability test on inflammatory cells showed that the differences were not statistically significant [p=.0864(middle); p=.7620 (posterior)].

Table 34 Distribution of epithelial cells in cytologic smears of conventional smokers.

	Anterior & Middle	Posterior	TOTAL
Nucleated	7	1 4	2 1
Non-nucleated	25	5	3 0
TOTAL	3 2	19	5 1

Table 35 Distribution of epithelial cells in cytologic smears of reverse smokers.

	Anterior	Middle	Posterior	TOTAL
Nucleated	18	7	2 4	49
Non-nucleated	5	23	16	44
Ghost	1	6	1	8
TOTAL	24	3 6	41	101

Table 36: Distribution of cytology slides according to micronuclei content (anterior area)

	none	few	plenty	Total
convenl	13	0	0	1 3
reverse	22	4	0	26
Total	3 5	4	0	39

Table 37: Distribution of cytology slides according to micronuclei content (middle area)

	none	few	plenty	Total
conven!	1 4	2	0	16
reverse	30	4	0	3 4
Total	44	6	0	50

Table 38: Distribution of cytology slides according to micronuclei content (posterior area)

,	none	few	plenty	Total
convenl	12	3	0	15
reverse	29	3	2	3 4
Total	41	6	2	4 9

Table 39: Distribution of slides according to smoking habit and presence of inflammatory cells(middle area).

	Reverse	Convenl	TOTAL
present	13	11	24
absent	21	6	27
TOTAL	3 4	17	5 1

Table 40 Distribution of slides according to smoking habit and presence of inflammatory cells(posterior area)

	Reverse	Convenl	TOTAL
present	2 4	11	3 5
absent	11	4	15
TOTAL	3 5	15	50

CHAPTER VI DISCUSSION

#### 6.0 DISCUSSION

### 6.1 Clinical features

The identification of high risk groups was first carried out in relation to medical problems that were observed to contribute to high mortality and morbidity rates (Beck, 1990). The process involves characterization of affected individuals and determination of common factors which may guide a clinician in predicting the occurrence of disease among individuals. These factors which epidemiologists refer to as risk factors, used to be identified by clinicians and/or researchers as they dealt with cases(Christie et al., 1987). To the clinician, establishing the role of risk factors in relation to ill-health has significance in differential diagnosis its and management. From an epidemiologist's stand-point, the ultimate goal of such an identification process has a broader scope. With the community in mind as a unit of study, it is recognized that this method can help in the planning of special that are aimed at prevention health programs and/or promotion of health status in identified groups.

These methods of identifying high risk groups can also be applied to dental conditions because of the inherent non-random distribution of most dental diseases(Beck, 1990). For example, it is now well recognized that benign and malignant oral mucosal changes have a high incidence in some countries where people practice various oral habits with and without the use of tobacco(Quigley et al., 1964; Morrow and Suarez, 1971, Pindborg et al., 1971; Gupta et al., 1980; Daftary et al., 1992).

Available data reveal the existence of varying disease patterns not only within subgroups but also between different countries (Waterhouse et al., 1982,; Binnie and Rankin ,1989; Whelan et al., 1990) which reflect socio-cultural characteristics and practices. For example in relation to oral cancer a comparison of incidence rates in different countries revealed that the incidence in Asia ranges from 1.6(for Japan) to a high 13.5 (India)(Daftary et al.,1992). For cancer incidence rates of different sites, Whelan et al(1990) reported that as for the lip, Southern Ireland has the highest incidence per 100,000 for males(11.6). Indian males had the highest incidence for the tongue(9.4) and France(13.5) for other sites of the mouth other than the lip, tongue and salivary glands. For females, Brazilians were reported to develop oral cancer at the highest rate(1.3) but not at as high a rate as male counterparts. Indian females dominate cancer rates for both tongue and other sites except the salivary glands. Interestingly, the Philippines ranks third to India and Singapore for both the tongue(ICD 141) and mouth(143-5) respectively. The same trend in ranking for females was shown by reports on the age-adjusted mortality rates for cancer of the oral cavity and pharynx (Binnie and Rankin, 1989).

Unpublished local studies in the Philippines reported the existence of 989 cases of cancer in different sites in 1970. Three-hundred and four of the cases(30.7%) were located in the oral cavity (review Eustaquio 1986). It was claimed that Filipino women are more susceptible to oral squamous cell

carcinoma of the palate and tongue than males(Eustaquio 1982, Mercado 1978, Amigo and Roman 1989).

This trend in morbidity and mortality rates in some Southeast Asian countries has been attributed to oral habits especially that of tobacco usage(Quigley et al., 1964; Mehta et al., 1969; Morrow and Suarez,1971; Bhonsle et al.,1976; Gupta et al.,1980; Daftary et al., 1992). Among tobacco habits, reverse smoking has been suspected (based on the local studies) as a strong risk factor associated with the development of oral cancer in the Philippines. Similar claims have been made by Pindborg et al.(1971), Reddy et al.(1973, Mehta et al.(1977), Gupta et al.(1980) and Gupta et al.(1986) for Indian subjects and by Morrow and colleague(1971) for Colombian subjects.

It has been suggested in the literature (Daftary et al.,1991) that oral cancer can be placed in the category of a preventable disease owing to:

- 1. Identification of aetiologic factors such as tobacco usage which can be controlled.
- 2. The ease of visual examination, where the dentist can play a substantial monitoring role.

However, Wright and Wright (1989) reflect on the fact that although the oral cavity is readily accessible to inspection and biopsy and that early, small lesions of oral cancer are curable, the attempt to improve oral cancer survival has not been

particularly successful. This is partly because medical and dental practitioners may not be aware of the true nature of premalignant and malignant oral lesions especially as they relate to various possible "intrinsic" and "extrinsic" factors which act together to produce neoplasms. (Binnie and Rankin 1989, Wright and Wright 1989, ) Moreover, although some lesions have been described as premalignant, the diagnosis of oral mucosal conditions relies heavily on the subjective interpretation of mucosal changes by the dentist (Malamos, Rosser and Scully 1986, Pindborg, Reibel and Holmstrup 1985). Furthermore in certain sectors of the population where accessibility to dental services is remote, it is a common practice that patients delay seeking professional advice until lesions reach an advanced stage when the prognosis is poor and therefore survival is lowered(Long 1984).

The view that the palatal changes associated with reverse smoking are probably precancerous mostly results from studies of Indian reverse chutta smokers in the districts of Srikakulam and Visakhapatnam, Andhra Pradesh (Reddy et al., 1973; Mehta et al.,1977; Gupta et al 1980). This view developed as a result of follow-up studies wherein a predominance of oral cancer cases was noted to occur among habitues of reverse smoking. In Srikakulam for example, it was observed in a 10-year follow-up study that the 11 new cases of oral cancer seen occurred solely in reverse smokers(Gupta et al.,1980). It was emphasized in these studies that palatal changes showed a varied clinical picture and histologic changes associated with a high percentage of epithelial dysplasia (Pindborg et al.,1971; Mehta et al.,1972).

In the later context, it was shown that of 381 palatal biopsies taken from Srikakulam subjects, 28% exhibited epithelial dysplasia. Mehta et al.(1977) reported a similar frequency (23%) from their study of 101 biopsies. In a separate study, Ramulu and Reddy(1973) found that out of 283 biopsies from female reverse smokers, 33% exhibited epithelial dysplasia. However, in some cultures such as the South Americans and Caribbeans and Indian Reverse dhumti smokers of Goa, India (mostly Catholic), epithelial dysplasia was not demonstrated(Bhonsle et al., 1976).

Due to the observed clinical variations exhibited by Indian subjects in the studies by Mehta and Gupta and colleagues, it was recommended that a distinction be made between the palate changes seen in reverse smokers and typical leukokeratosis nicotina palati as is observed among pipe and cigar smokers of Western countries. Based on the findings of the present study, it appears that the observed palatal mucosal changes in Filipino women reverse smokers, though not as diverse as those seen in Indian subjects, also show considerable variation with features that do not fall within the classic description of stomatitis nicotina observed in pipe and cigar smokers of Western countries.

From the clinical examination of subjects in this study, three main groups of palatal changes were identified:

Group 1: the palatal mucosa only exhibited areas of intramucosal brown-black pigmentation.

(Figures 10 - 11)

- Group 2: the palatal mucosa presented a combination of changes including leukoplakia, mucosal thickening, fissuring, nodularity, staining and erythema. Some subjects presented enlargement and reddening of the orifices of minor salivary gland ducts. (Figures 12 43)
- Group 3: the mucosa presented variable ulceration, severe reddening and nondescript roughening.

  (Figure 44 45)

The clinical palatal changes observed in the majority (Group 2) of reverse smoking subjects in this study as a group exhibited some of the changes described in Indian reverse smokers by Mehta and colleagues(1977) and Gupta et al(1980) However, in present study the distinct nodularity described by Mehta et al(1977) was not a prevalent finding. In the majority of subjects the predominant change noted was thickening and fissuring of palatal epithelium usually associated with superficial yellow-brown discoloration. Based on the clinical changes observed, it was deemed inappropriate to classify the lesions into the categories described by Mehta and co-workers(1977). The preceding classification of palatal mucosal changes in Filipino women smokers was therefore based on the combination of features present and their extent or severity.

## 6.2 Demographic and habit variables

The palatal changes observed in the present study also differ from those described in Caribbean and South American subjects(Quigley et al 1964, Quigley et al 1966). These observational differences reinforce the view that variables related to the tobacco product used for reverse smoking and to other as yet undefined variables including age, gender, frequency and duration of smoking and whether the habit is practiced in association with other tobacco habits (e.g. conventional smoking, chewing, snuff usage, pan(betel) usage) may be important in determining the nature of clinical changes The results of analysis on confounding and observed. interaction of four demographic and habit variables (age, educational level, use of filtered versus non-filtered cigarette, duration of smoking) examined in this study gave results that support this view. The inequality of the adjusted Odds Ratios for all factors at different levels to the crude Odds Ratio confirms a confounding effect of the four variables. As can be seen from the results in Tables 4, 8, 15 and 19, there was either an over-estimate or underestimate of the true effect of the smoking habit by the crude Odds Ratio shown in Table 13. Hence, a strong or weak association could be concluded on the basis of the crude estimate alone. These results imply to a certain degree that indeed, environmental factors act in combination to produce changes and that variations in effect occur as a result of interaction and/or co-action of factors.

In addition to environmental factors, considerable attention had been given to indicators of social class or socioeconomic status because of their association with risk factors and diseases (Marmot, Kogevinas and Elston, 1987). This is based on the premise that social class incorporates economic, political and cultural differences that may have an impact on health.

Variations in disease rates by scrutiny of socio-cultural factors had been recognized to give a better understanding of the components of the causal chain between social position and disease risk (Marmot, Kogevinas and Elston, 1987). Liberatos and colleagues (1988) proposed that if a study aims to find out the relationship of socio-cultural factors and disease, socioeconomic status is one factor that needs consideration because it has been claimed that poor measurement of social class leading to random misclassification will dilute any actual bivariate associations. It is important that careful selection of indicators of social class are used because misleading results may be obtained if inappropriate factors are chosen. Antonovsky (1967) for example noted that persons in lower social strata have been found to have lower life expectancy and higher mortality rates from all causes of death combined. Bassett and Krieger (1986) recently reported that the lower survival in black as compared with white breast cancer patients could largely be explained by the lower social class of blacks, since the association between race and survival disappeared when social class was controlled for. Hence, social class can also be considered as a starting point for providing clues about

many aetiologic agents. Though the sample of the present study is limited, a significant finding for some variables can give direction to future studies encompassing larger populations.

The development of measures of social class in order to quantify relative position within society were mostly initiated by sociologists (Liberatos et al., 1988). Max Weber held the view that differential societal position is based on three dimensions: social class, status and party (Weber, 1946). Class is assumed to have an economic base. It implies ownership and control of resources and is indicated by measures of income (Lipset, 1968). Status is considered to be prestige or honour in the community as it implies "access to life chances" if based on social and cultural factors such as family background, lifestyle and social networks (Weber, 1946; Lipset 1968). Lastly, power is taken in relation to its political context.

In the literature (Haug, 1977; Hauser and Featherman, 1977; Nam and Terrie, 1982), three outstanding indicators of social class are described:

- a) Occupation
- b) Education
- c) Income or wealth
- a) Occupation Many sociologists feel that occupation is a reliable single indicator of relative standing in industrial societies (Haug, 1977). It is considered to be related to differential exposure to a physically noxious or

psychologically stressful environment and to the degree of intrinsic and extrinsic rewards, job security and personal control in the work environment. It is also considered to reflect access to medical care and the ability to obtain good housing (Zurayk, Halabi and Deeb, 1987; Marmot, Kogevinas and Elston, 1987).

- Education is the second indicator which some authors b) consider as a better indicator of socio-economic status (Wynder and Stellman, 1977). This indicator is considered to be related to health outcomes through its influence on lifestyle behaviours (e.g. exercise, diet) (Chirikos, Reiches and Moeschberger, 1984; Jacobsen and Thelle, 1988), problem-solving capacity and values (e.g. importance of preventive health behaviours) (Marmot, et al., 1987; Zurayket al., 1987). Education is sometimes assumed to be a precursor to income and occupational attainment (Hauser and Featherman, 1977). However, it must be emphasized that high education does not necessarily lead to high income and high occupational standing.
- c) Income or wealth the third indicator has its effect on opportunities for education and provides access to different lifestyles, prestige or power. (Barber, 1968). Furthermore, income is considered to provide access to medical care resources and good housing, less exposure to a noxious environment and a good diet, good working

conditions and more social amenities (Marmot, et al., 1987; Zurayk et al., 1987).

In the present study, of the three indicators described above, only educational level was found to affect disease rate estimates. It must be noted that all reverse smokers encountered were elderly women most of whom had not reached beyond level four of primary education. This has some implications on the reasons given by the women for perpetuation of the habit, namely:

- a) a belief that the warmth from exhaled smoke can relieve the pains of colic in a newborn infant.
- b) to drive mosquitoes away while gathering sweet potatoes from the forest.
- to keep the body warm while in rice fields during the planting season.
- d) to prevent the ashes of the cigarette from falling onto clothes while washing.
- e) to neutralize the smell of soiled diapers of newborn babies while washing diapers.

Because of the women's strong belief in the soothing effect of reverse smoking, the habit is perpetuated and addiction follows. For most of the Filipina habitues reverse smoking reportedly provided pleasure similar to food intake. Although some of the women were aware of the changes occurring in their palatal

mucosa, the effects were not considered alarming or sufficient worrisome to stop them from practicing the habit.

## 6.3 Exfoliative cytology

The value of exfoliative cytology in assessing the status of mucosal lesions has been explored (Mehta et al., 1972). application of exfoliative cytology as a morphological method for detecting neoplastic condition of the mucosa was based on its success in the early detection of uterine cervical cancer (Bernstein and Miller, 1978; Chomette et al., 1980). The popularity of this diagnostic technique lies on the fact that large areas can be screened in less time and repetition is virtually unlimited because no surgical procedure is needed. (Burkhardt, In a study population where the subjects are apprehensive to undergo biopsy procedures, exfoliative cytology is one screening procedure which could be used for superficial microscopic assessment of affected tissues. However, because of the absence of a transformation zone that can exhibit squamous metaplasia, as in the uterine cervix, reliability of exfoliative cytology for oral cavity lesions has been considered This view is supported by high false-negative results of low. 31%, 37% or even 62% in some studies (Folsom et al., 1972; Reddy et al., 1975; Zallen, 1978).

The usefulness of the micronucleus test gained popularity as a reliable screening procedure in vivo for detecting chemically induced structural and numerical chromosome aberrations on bone marrow cells of mice (Matter and

Grauwiler, 1974; Schmid, 1975). The usefulness of this test, like other short-term test systems, is based on fullfilment of certain The tests used must screen for the types of requirements. genetic damage that are generally considered to be the cause of human disorders. They should further provide for mammalian metabolism. Added to this are economic and time requirements which must not be prohibitive. Stich and Rosin (1983) suggested that the application of the micronucleus test to human exfoliated cells could provide evidence for carcinogeninduced cytogenetic damage and might therefore reflect the carcinogenic hazard in a particular organ or tissue. frequency of micronuclei in exfoliated cells can therefore be used as an "endogenous dosimeter" present in the tissue which is the target for carcinogenic agents and from which carcinomas arise. The present study explored the possibility of using the micronucleus test in assessment of palatal cytology in reverse smoking subjects. The application of this test to exfoliated cells of the palate in the present study revealed a negative result largely because of keratohyalin granules which can be found in the epithelial cells of the palate could be mistaken for micronuclei.

The results of the present study indicate that no significant difference in cytologic features could be detected between the two study groups in terms of predominant cytologic features observed. This implies that the usefulness of cytology in the screening of palatal lesions in Filipino reverse smokers should be addressed in future, larger studies. However, as the palatal mucosal epithelium of Filipino reverse

smokers shows gross clinical thickening the value of superficial exfoliative cytology can probably be questioned. Of greater value would be incisional biopsy material.

CHAPTER VII

CONCLUSIONS

#### 7.0 CONCLUSIONS

The description of observed clinical palatal changes in reverse smoking and conventional smoking subjects in this study showed that 96.7% ( 59/61 ) reverse smokers exhibited mucosal change while 26.7% ( 8/23 ) of conventional smokers exhibited evidence of palatal mucosal alteration chiefly pigmentation. Whether the later was acquired or intrinsic ( i.e., racial ) could not be determined.

The palatal changes among reverse smokers included leukoplakia, staining, fissuring and thickening of the mucosa. Increased prominence of salivary gland duct openings was observed in some subjects and palatal nodularities were seen in a proportion of subjects. Variation in the severity and distribution of changes was observed with the majority of subjects exhibiting moderate change in a scale of mild-moderate-severe.

On the basis of analysis of the clinical features seen it was concluded that the range of changes seen which incorporating some features stomatitis nicotina did not correspond to classic description of this entity.

Analysis of cytologic smears from both reverse and conventional smokers failed to reveal significant difference in cell populations present on smears. An attempt to identify micronuclei in palatal smears from reverse smokers was not successful.

One of the purpose of this study addressed the possible role of demographic and habit variables in identifying Filipino women who may have a higher probability of exhibiting palatal mucosal changes given that they preferred to smoke in one in two ways(reverse smoking or conventional smoking). Of the different risk factors considered in the identification process, subjects of the two study groups differed significantly with respect to two of the seven demographic variables and two of the ten habit variables.

It must be recognized that the greatest limitation in this study which may have resulted in weak associations detected with other variables is the possibility of information bias in data gathered. Information bias in the present study may be due to:

- a) restricted sample size
- b) inability of subjects to recall precise information
- c) inadvertent under or over declaration by the subjects

In addition, the voluntary nature of participation into the study posed a considerable restriction to the external validity of results.

Despite the limitations mentioned, this study showed that:

- 1) Reverse smoking is predominantly practiced by elderly Filipino women.
- 2) The habit is perpetuated mainly because of a belief that smoker can have a soothing effect on babies.
- 3) More reverse smokers had less schooling than conventional smokers.
- 4) Non-filtered cigarettes are preferred by reverse smokers while filtered cigarettes are preferred by conventional smokers.
- 5) Reverse smokers, in general, have a longer history of smoking than conventional smokers.
- There is a tendency for both confounding and interaction of effects by four significant variables namely age, educational attainment, use of filtered versus non-filtered cigarettes and duration of smoking.

Although it is recognized that the results of this study may not reflect the whole population of Filipina smokers in general, it is hoped that it provides a better framework for modelling future studies aimed at establishing a risk assessment model for precancerous and/or cancerous oral lesions and reverse smoking.

REFERENCES AND APPENDICES

### REFERENCES

- AMIGO, J.A. AND ROMAN, A. (1990): A retrospective study on the prevalence of oral cancer among smoking patients examined at The Philippines General Hospital from the years 1979 - 1989.
- ANTONOVSKY, A.(1967): Social class, life expectancy and overall mortality. Milbank Mem Fund Q, 45: 31 73.
- ARMSTRONG, B and DOLL R. (1975): Environmental factors and cancer incidence and mortality in different countries with special reference to dietary practices. Int. J. Cancer, 15: 617 631.
- AXELL, T. (1976): A prevalence study of oral mucosal lesions in an adult Swedish population. Odontol. Revy. 27, Suppl. 36:1-103.
- AXELL, T., HOLMSTRUP, P., KRAMER, I.R.H., PINDBORG, J.J. and SHEAR, M. (1984): International seminar on oral leukoplakia and associated lesions related to tobacco habits. Community Dent. Oral Epidemiol., 12: 145.
- AVERY, J.K. (1987): Oral Development and Histology, Williams and Wilkins, Baltimore, USA, pp. 206-220.
- BANOCZY, J. (1982): Oral Leukoplakia.

  Martinus Nijhoff Publishers, Netherlands and Akademiai Kiado, Hungary.
- BARBER, B. (1968): Introduction to social stratification. Int. Encycloped. Soc. Sci. 15: 288-298.
- BASSETT, M. T. and KRIEGER N (1986): Social class and blackwhite differences in breast cancer survival. Am. J. Public Health, 76:1400-3

- BASTIAAN, R. J. and READE, P. C.(1980): The histopathologic features which follow repeated applications of tobacco tar to rat lip mucosa. Oral Surg. Oral Med. Oral Pathol. 49:435-440.
- BECK, J. D. (1990): Identification on risk factors. in: Bader, J.D., ed. Risk assessment in dentistry, Chapel Hill, NC: University of North Carolina, pp. 8-13.
- BERNSTEIN, M.L. and MILLER, R.L. (1978): Oral exfoliative cytology. J Am. Dent. Assoc., 96:625.
- BHONSLE, R.B., MURTI, P.R., GUPTA, P.C., AND MEHTA, F.S. (1976): Reverse dhumti smoking in Goa: an epidemiologic study of 5449 villagers for oral precancerous lesions. Ind. J. Cancer, 13: 302-305.
- BINNIE, W.A., RANKIN, K.V. (1984): Epidemiological and diagnostic aspects of oral squamous cell carcinoma.

  J. Oral Pathol., 13: 333-341.
- BINNIE, W.A., RANKIN, K.V. and MACKENZIE (1983): Etiology of oral squamous cell carcinoma.

  J. Oral Pathol., 12:11-29.
- BINNIE, W.H. and RANKIN, K.V. (1989): Epidemiology of Oral Cancer In: Wright, B.A., Wright, J.M. and Binnie, W.H. eds. Oral Cancer: Clinical and Pathological Considerations. CRC Press Inc., Boca Raton, Florida. pp. 1 10.
- BLOT, W.J., McLAUGHLIN, J.K., WINN, D.M., AUSTIN, D.F., GREEBERG, S., PRESTON-MARTIN, S., BERSTEIN, L., SCHOENBERG, J. B., STEMHAGEN, A., FRAUMENI, J. F. Sr. (1988): Smoking and drinking in relation to oral and pharyngeal cancer. Cancer Res., 48: 3282 -3287.

- BONETT, A., RODER, D. (1987): Epidemiology of Cancer in South Australia: Incidence, mortality and survival, 1977-1985. Incidence and mortality, 1985. Analyzed by type, country of birth and geographic location. Adelaide: Lutheran Publishing House.
- BRUCE, W.R., HEDDLE, J.A. (1979): The mutagenic activity of 61 different agents as determined by the micronucleus, salmonella and sperm abnormality assays.

  Can. J. Genet. Cytol. 21: 319-334.
- BURKHARDT, A (1985): Advanced methods in the evaluation of premalignant lesions and carcinomas of the oral mucosa. J. Oral. Pathol. 14:751 -778.
- BURKHARDT, A. (1987): Oral cavity and oropharynx in "Diseases of the head and neck. An atlas of histopathology. Arnold, W.J., Laissue, J. A., Friedman, I., Naumann, H.H. (editors). Thieme Medical Publishers, Inc. New York. 6.12 6.20.
- CHAING CL (1968): Introduction to stochastic processes in biostatistics. New York: Wiley.
- CHIRIKOS, T. N., REICHES, N. A., MOESCHBERGER, M. L. (1984):

  Economic differentials in cancer survival: a

  multivariate analysis. J. Chronic Dis., 37:183 -193.
- CHOMETTE, G., AURIEL, M, SZPIRGLAS, H. (1980): Cytologie exfoliative de la cavite buccale valeur pour le detection et la controle therapeutique des cancers de la cavite buccale. Arch Anat Cytol Pathol. 28: 169-173.
- CHRISTEN, A.G. AND GLOVER, E.D. (1981): Smokeless tobacco: seduction of youth. World smoking and Health. 6:20 24.

- CHRISTEN, A.G., SWANSON, B.Z., GLOVER, E. D., and HENDERSON, A. H. (1982); Smokeless tobacco: The folklore and social history of snugging, sneezing, dipping and chewing. J. Am. Dent. Assoc., 105: 821-829.
- CHRISTEN AG, McDONALD JL, CHRISTEN JA (1991): The impact of tobacco use and cessation on nonmalignant and precancerous oral and dental diseases and conditions. An Indiana University School of Dentistry Teaching monograph, Indianapolis, Indiana.
- CHRISTIE, D., GORDON, I., and HELLER, R. (1987): An introductory text for medical and other health science students. New South Wales University Press, NSW, Australia
- DABELSTEEN E, ROED-PETERSEN B, SMITH J and PINDBORG JJ (1971): The limitation of exfoliative cytology for the detection of epithelial atypia in oral leukoplakias. Brit. J. Cancer, 25: 21 24.
- DAFTARY DK, MURTY PR, BHONSLE RB, GUPTA PC, MEHTA FS and PINDBORG JJ (1992): Oral precancerous lesions and conditions of tropical interest. In Oral diseases in the tropics; Prabhu SR, Wilson DF, Daftary DK and Johnson NW, Eds., Chapter 36:402 428.
- DAFTARY, D.K., MEHTA, F.S., GUPTA, P.C. and PINBORG, S.S. (1972):The presence of candida in 723 oral leukoplakias among Indian villagers.

  Scand. J. Dent. Res., 80:75-79.
- DOLL R (1977): Strategy for detection of cancer hazards to man. Nature, 265:589 - 596.
- DOLL, R., PETO, R. (1981): The causes of cancer Quantitative estimates of available risks of cancer in The United States today. Oxford University Press.

- DANIEL, W.W. (1983): Biostatistics: A foundation for Analysis in the Health Sciences, third edition, New York, Wiley.
- ELWOOD, J.M., PEARSON, J.C.G., SKIPPEN, D.H., and JACKSON, S.M. (1984): Alcohol, smoking, social and occupational factors in the etiology of cancer of the oral cavity, pharynx and larynx. Int. J. Cancer, 34: 603-612
- EUSTAQUIO, R. (1986): Comparative study on the prevalence of oral cancer for periods 1973 1976 and 1980 1983, a postgraduate thesis paper submitted to the College of Public Health, University of the Philippines Manila.
- EVANS AS and NIEDERMAN JC(1984): Epstein-Barr virus. In: Evans AS, ed. Viral infection of humans: Epidemiology and control. New York, Plenum Press.
- FOLSOM TC, WHITE CP, BROMER L, CANBY HF, GARRINGTON G (1972): Oral exfoliative study. Oral Surgery, 33(1): 61 72.
- FRANK DW, TROZS RJ and GOOD PI (1978): A comparison of two methods for evaluating drug-induced chromosomal alterations. Mutation Res., 56:311 317.
- GRAHAM S and REEDER LG(1979): Social epidemiology of chronic diseases. In: Freeman H, Levine S, Reeder LG eds. Handbook of medical sociology. Englewood Cliffs, NJ: Prentice-Hall.
- GUPTA PC, MEHTA FS, DAFTARY DK, PINDBORG JJ, BHONSLE RB, JALNAWALLA PN, SINOR PN, PITKAR VK, MURTI PR, IRANI RR, SHAH HT, KADAM PM, IYER KSS, IYER HM, HEDGE AK, CHANDRASHEKAR GK, SHROFF BC, SAHIAR BE and MEHTA MN (1980): Incidence rates of oral cancer and natural history of precancerous lesions in a 10-year follow-up study of Indian villagers. Community Dental Oral Epidemiol., 8:287 333.

- GUPTA PC, MEHTA FS, PINDBORG JJ (1984): Mortality among reverse chutta smokers in South India. Br. Med. J. 289:865 866.
- GUPTA PS, BHONSLE RB, MEHTA FS and PINDBORG JJ (1984):

  Mortality experience in relation to tobacco chewing and smoking habits from a 10-year follow-up study in Ernakulam district, Kerala. Inte. J. Epidem. 13: 184-187.
- GUPTA PC, PINDBORG JJ, BHONSLE RB, MURTI PR, MEHTA FS, AGHI MB, DAFTARY DK, SHAH HT and SINOR PN (1986): Intervention study for primary prevention of oral cancer among 36,000 Indian tobacco users. Lancet. 1235 1239.
- HARDY JD (1956): The nature of pain. J. Chron. Dis., 4: 22 51.
- HAUG MR (1977): Measurement in social stratification. Annu Rev Sociol., 3: 51 77.
- HAUSER RM, FEATHERMAN DL (1977): The process of statification: trends and analyses. New York: Academic Press.
- HEDDLE JA (1973): A rapid in vivo test for chromosomal damage, Mutation Res., 29: 89 96.
- HIGGINSON J(1976): In environment pollution and carcinogenic risk. IARC scientific publication No. 13, Paris, INSERM.
- HOFFMAN D, RATHKAMP G and WYNDER EL(1963): Comparison of the yields of several selected components in the smoke from different tobacco products. J. Natl. Cancer Inst., 31: 627 637.

- HOGEWIND WF, GREEBE RB and van der WAAL I(1987): Reverse smoking in the Netherlands. Int. J. Oral Maxillofacial Surg., 16(4): 500 4.
- IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans (1985): Tobacco habits other than smoking; Betelquid and Areca- Nut Chewing, Some Related Nitrosamines, Vol. 37. Lyons: International Agency for Research on Cancer.
- IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans; Tobacco Smoking, Bol. 38. Lyons: International Agency for Research on Cancer.
- JACOBSEN BK, THELLE DS(1988): Risk factors for coronary heart disease and level of education. The Tromso Heart Study. Am. J. Epidemiol., 127:923 932.
- KANE P(1991): Women's health from womb to tomb. Macmillan Press, London. Ch 1 4, 1-108.
- KELSEY JL, THOMPSON WD, EVANS AS (1986): Methods in observational epidemiology. Oxford University Press.
- KLEINBAUM DG, KUPPER LL and MORGENSTERN H (1982): Epidemiologic research: principles and quantitative methods. Belmont, CA: Lifetime Learning Publications, 1982.
- LIBERATOS P, LINK BG AND KELSEY JL (1988): The measurement of social class in epidemiology. Epidemiol Rev.10:87 121.
- LIND PO (1987): Malignant transformation in oral leukoplakia. Scand. J. Dent. Res. 95: 449 - 455.
- LIPSET SM (1968): Social class. Int. Encyloped. Soc. Sci., 15: 298
   316.

- LONG AF (1984): Research into health and illness issues in design, analysis and practice.
- MAHBOUBI E and SAYED GM (1982): Oral Cavity and Pharynx, In: D. Schottenfeld and J.F. Fraumeni, Jr., (eds), Cancer epidemiology and prevention, pp. 583 - 595. Philadelphia: WB Saunders Co.
- MALAMOS, D. PRIME, S.S., ROSSER, T. AND SCULLY, C.(1986): Oral epithelial atypia and acantholytic dyskeratosis in rats painted with 4-nitroquinoline-N-oxide. J. Oral Pathol. 15:280 283.
- MARMOT MG, KOGEVINAS M and ELSTON MA (1987): Social/economic status and disease. Annu. Rev. Public Health, 8:111-35.
- MATTER BE and GRAUWILER J (1974): Micronuclei in mouse bone-marrow cells. A simple in vivo model for the evaluation of drug-induced chromosomal aberrations. Mut. Res., 23:239 249.
- MATTER BE, JAEGER I and GRAUWILER J (1974): The relationship between the doses of various chemical mutagens required to induce micronuclei in mouse bone marrow and their lethal dose, Proc. Eur. Soc Study Drug Toxicity, Vol. 14, Excerptea Medica, Amsterdam,ICS No. 311 pp. 303 307.
- MATTER BE and SCHMID W (1971): Trenimon- induced chromosomal damage in bone marrow cells of six mammalian species, evaluated by the micronucleus test.

  Mut. Res. 12: 417 425.
- MEHTA FS; PINDBORG JJ and GUPTA PC (1969): Epidemiologic and histologic study of oral cancer and leukoplakia among 50,915 villagers in India. Cancer, 24: 832 849.

- MEHTA FS, SAHIAR BE, DAFTARY DK, GUPTA PC and PINDBORG JJ (1972): A correlative histocytological study of carcinoma and epithelial atypia of the palate among Indian reverse smokers. Br. J. Cancer, 26: 230 233.
- MEHTA FS, JALNAWALLA PN, DAFTARY DK, GUPTA PC and PINDBORG JJ (1977): Reverse smoking in Andhra Pradesh, India: variability of clinical and histologic appearances of palatal changes. Int. J. Oral Surg., 6: 75 83.
- MEHTA FS, AGHI MB, GUPTA PC, PINDBORG JJ BHONSLE RB, JALNAWALLA PN nad SINOR PN (1982): An intervention study of oral cancer and precancer in rural Indian populations: a preliminary report. Bulletin of World Health Organization, 60(3): 441 446.
- MERLETTI F, BOFFETTA P, GIOVANNINO C, MASHBERG A and TERRACINI B (1989): Role of tobacco and alcoholic beverages in the etiology of cancer of the oral cavity/oropharynx in Torino, Italy. Cancer Res. 49: 4919 4924.
- MIETTINEN OS (1976): Estimability and estimation in casereferent studies. Am. J. Epidemiol., 103:226 - 35.
- MIETTINEN OS(1985): Theoretical epidemiology principles of occurrence research in Medicine. John Wiley and sons. New York.
- MILTON, J.S. (1983): Statistical Methods in the Biological and Health Sciences. New York: Mc Graw-Hill.
- MOORE C and CATLIN D (1967): Anatomic origins and locations of oral cancer. Amer. Jour. Surg., 114: 510 513.

- MORGENSTERN J, KLEINBAUM DG and KUPPER L (1980): Measures of disease incidence used in epidemiologic research. Internat. J. Epidemiol, 9: 97 104.
- MORROW RC and SUAREZ G (1971): Mucosal changes and cancer in intraoral smoking. Laryngoscope, 81:1020 1031.
- NAM CB and TERRIE EW (1982): measurement of socioeconomic status from United States census data. In: Powers MG, ed. Measure of socio-economic status: current issues. Boulder, CO: Westview Press.
- PINDBORG JJ, MEHTA FS, GUPTA PC, DAFTARY DF and SMITH CJ (1971): Reverse smoking in Andhra Pradesh, India: A study of palatal lesions among 10,169 villagers. The Br. J. Cancer, 25:10 20.
- PINDBORG JJ (1980): Oral Cancer and Precancer. John Wright and Sons Ltd., Triangle West, Bristol, 42 -44.
- QUIGLEY LF, COBB CM, SCHOENFELD S, HUNT EE and WILLIAMS P (1964): Reverse smoking and its oral consequences in Caribbean and South American peoples. J. Am. Dent. Assoc, 69: 426 442.
- QUIGLEY LF, COBB CM and HUNT EE (1965): Measurement of oral and burning zone temperatures during conventional and reverse cigarette smoking. Arch oral Biol., 10: 35 44.
- QUIGLEY LF, SHKLAR G and COBB CM (1966): Reverse cigarette smoking in Caribbeans: Clinical, histologic and cytologic observations. J. Am. Dent. Assoc., 72: 867 973.
- RAMULU C and REDDY CRRM (1973): Pattern of changes produced in the palates of female reverse smokers of chuttas. Indian J. Cancer, 10: 191-198.

- REDDY CRRM, KAMESWARI VR, RAMULU C and REDDY PG(1971): Histopathological study of stomatitis nicotina. Br. J. Cancer, 25: 403 410.
- REDDY CRRM, RAJU MVS, RAMULU C and REDDY PG (1972): Changes in the ducts of the glands of the hard palate in reverse smokers. Cancer, 30: 231 238.
- REDDY CRRM, RAMULU C, RAJU MVS, SUNDARESHWAR B, RAM MURTHY B, SASTRY B and NARASIMHAM R (1973): Preliminary report on the epidemiology of reverse smoking and palatal precancerous lesions in the rural population of Visakhapatnam district. Indian J Med Res., 61: 838 847.
- SCHLESSELMAN JJ (1982): Case-control studies: design, conduct, analysis. New York: Oxford University Press.
- SCHMID W (1973): Chemical mutagen testing on in vivo somatic mammalian cells. Agents and actions, 3: 77 85.
- SCHMID W (1975): The micronucleus test. Mut. Res., 31: 9-15.
- SCHWARTZ DL (1965): Stomatitis nicotina of the palate, Oral Surg. Oral Med. Oral Pathol., 20: 306 315.
- SELVIN S (1991): Statistical Analysis of Epidemiologic data. Monographs in epidemiology and biostatistics. Vol 17. Oxford Univ. Press, N.Y.
- SHIROKOV EP (1960): Carcinoma of the palate. Am J. Surg., 100: 530.
- SILVERMAN S (1985): Oral Cancer. 2nd edition, The American Cancer Society, New York.
- STEVENS, B.C. (1976): The collector's book of snuff bottles. New York, John Weatherhill Inc., pp.1 312.

- STICH HF and ROSIN MP (1983): Micronuclei in exfoliated human cells as an internal dosimeter for exposures to carcinogens. In: H.F. Stich(ed.), Carcinogens and mutagens in the environment, Vol II, Naturally occurring compounds: endogenous formation and modulation, CRC Press, Boca Raton, Florida.
- SUSSER M, WATSON W and HOPPER K(1985): Sociology in Medicine. New York: Oxford University Press.
- THOMA KH (1941): Stomatitis nicotina and its effect on the palate. Am. J. Orthod. (Oral Surg), 27: 38-47.
- TORO L and BRUNICARDI AS (1991): Carcinoma espinocellular del paladar. Acta Odontologica Venezolana. 29(2): 60 64.
- VAN WYKE CW (1967): Nicotina stomatitis of the palate a clino-histological study, J. Dent., Assoc., South Africa, 22: 106 111.
- VOGES E (1984): Tobacco encyclopedia, Mainz, Tobacco Journal International.
- WATERHOUSE, J. MUIR, C., SHANMUGARATNAM, K. AND POWELL, J.(EDS)(1982): Cancer incidence in five continents. Vol. IV, International Agency for Research on Cancer, World Health Organization, Lyon, France.
- WEBER M (1946): Class, status and party. In; Gerth H, Mills CW, eds. From Max Weber: essays in Sociology. New York: Oxford University Press
- WHELAN SL, PARKIN DM, and MASUYER E (1990): Patterns of cancer in five continents; International Agency for Research on Cancer, World Health Organization, Lyon, France.

- WILSON DF, GRAPPIN G and MIGUEL JL (1992): Traditional, cultural and ritual practices involving the teeth and orofacial soft tissues. In: Oral diseases in the tropics, Prabhu SR, Wilson DF, Daftary DK and Johnson NW (eds.), Chapter 9: 103 119.
- WINN DM, ZIEGLER RG, PICKLE LW, GRIDLEY G, BLOT WJ and HOOVER RN(1984): Diet in the etiology of oral and pharyngeal cancer among women from the southern United States. Cancer Res., 44: 1216 1222.
- WHO Collaborating Centre for Oral Precancerous Lesions (1978):

  Definition of Leukoplakia and related lesions An aid to studies on Oral Precancer, Oral Surg. Oral Med. Oral Pathol. 46(4): 518 539.
- WYNDER EL, MUSHINSKI MH and SPIVAK JC (1977): Tobacco and alcohol consumption in relation to the development of multiple primary cancers. Cancer, 30:1872 78.
- WYNDER, E.L., COVEY, L.S., MABUCHI, K. and MUSHINSKI, M. (1976): Environmental Factors In Cancer of the Larynx. Cancer, 38: 1591 1600.
- WYNDER, E.L. MUSHINSKI, M.H. and SPIVAK, J.C.(1977): Tobacco and alcohol consumption in relation to the development of multiple primary cancers. Cancer, 40: 1872 78.
- WYNDER EL and STELLMAN SD(1977): Comparative epidemiology of tobacco related cancers. Cancer Res., 37: 4608 -22.
- ZURAYK H, HALABI S and DEEB M(1987): Measures of social class based on education for use in health studies in developing countries. J. Epidemiol. Comm. Health. 41: 173 -9.

## **APPENDICES**

APPENDIX ONE :

Flow of Study

APPENDIX TWO

Consent Form

APPENDIX THREE :

Sample Size Estimation

APPENDIX FOUR

Survey Questionnaire

APPENDIX FIVE

Codelist for Data entry

#### APPENDIX 1

# Flow Diagram of the study

As outlined by Kleinbaum et al., (1982), an observational study of the cross-sectional type has the following sequence of events:

 $\mathbf{E}$ 

N --- s ---

 $\mathbf{E}$ 

### where:

- N = refers to target population or all women smokers in the Philippines.
- C = refers to prevalent cases or those subjects exhibiting palatal mucosal changes.
- C = refers to noncases or subjects exhibiting relatively normal appearing mucosa.
- E = refers to subjects who are exposed to the possible aetiologic factor under study which is the practice of the habit of reverse smoking.
- E = refers to subjects who engage in conventional smoking.

# THE UNIVERSITY OF ADELAIDE CONSENT FORM (ULAT PAKIKIPAGSANG-AYON)

1.	Iplease print) hereby consent to take  (Ako si ay sumasang-ayon na mahibahagi sa  part in the research project entitled "A Pilot Study on Reverse Smoking  Practices Among Filipinos".  (proyektong "A Pilot Study on Reverse Smoking Practices Among Filipinos".)			
2.	I have had the project, so far as it affects me, fully explained to my (Ang proyektong ito ay naipaliwanag na mabuti sa akin ng sumasagawa ng satisfaction by the research worker. My consent is given freely. (pagsusuri. Ang pagsang-ayon ko ay bukal sa aking kalooban).			
3.	Although I understand that the purpose of this research project is to (Bagama't naiintindihan kong ang layunin ng pagsusuring ito ay ang mapabuti ang improve the quality of dental care, it has also been explained that my (pangangalaga sa kalusugan ukol sa bibig, naipaliwanag din sa akin na ang involvement may not be of any benefit to me. pakikibahagi ko ay maaring walang maibubuti para sa akin.).			
4.	I have been given the opportunity to have a member of my family or a (Ako ay binigyan ng kaukulang kalayaan upang isa sa mga kaanak o kaibigan ko friend present while the project was explained to me. ay makasama ko habang ang pagsusuring ito ay ipinapaliwanag sa akin.)			
5.	I have been informed that while information gained during the study may (Ipinaalam sa akin na bagama't mapapalathala ang kaalamang maidadagdag sa be published, I will not be identified and my personal results will not be pagsusuring ito, ang psgkatao ko ay hindi ipapahayag). divulged.			
6.	I understand that I am free to withdraw from the project at any time and (Naiintindihan kong ako ay may kalayaang tumiwalag sa proyekto ano mang this will not affect dental advice in the management of my oral oras at ito ay hindi makaka-apekto ng pangangalaga ng kalusugan ko ngayon at sa health, now or in the future.  hinaharap).			
7.	I am aware that I should retain a copy of this Consent Form when (Maliwanag sa akin na pagkatapos ng ulat na ito, dapat akong magkaroon completed.  ng kopya ng pagsang-ayon na ito).			
Signed	Signed Date			
(Lagda) (Petsa)				
Name	of WitnessSigned			

(Lagda)

(Petsa)

Date .....

(Pangalan ng saksi)

Ι	. have described to
(Ako si	ay ipinaliwanag kay
the nature of the procedures to be carrang mga isasagawang pagsusuri. Sa a the explanation. paliwanag.	ried out . In my opinion she/he understood king kaalaman, naiintindihan niya ang
SIGNED	DATE
(LAGDA)	( PETSA)
STATUS IN PROJECT	
( KATUNGKULAN SA PROYEKTO)	

## Sample Size Estimation

Conditions assumed:

- a) no confounders
- b) one per case
- c) equal costs

Formula:

$$n = \frac{[Z \propto pq + Z_B p_1 q_1 + p_0 q_0]^2}{[p_1 - p_0]^2}$$

where:

- po exposure rate among controls (or the general population if the disease is rare). In this study, this was estimated by the proportion of reverse smokers among subjects who did not have mucosal change.
- R is the relative risk to be detected

$$p_0 R$$
 $\frac{p_0 R}{1 + p_0 (R - 1)}$ 

$$p = \frac{p_1 + p_0}{2}$$

n - number of subjects per group

On the basis of the first 74 subjects, the following computations were derived:

$$p_0 = 5/19 = .3$$

$$R = 91/.33 = 2.75 3$$

$$p_1 = (.3)(3) = .56 : q_1 = .1 - .56 = .44$$
  
  $1 + .3(3-1)$ 

$$p = .56 + .3 = .43$$
  $q = .57$ 

$$n = \underbrace{[1.96 \quad (.43)(.57) + 1.64 \quad (.2464) + (.21)]^2}_{(.56 - .3)^2}$$

$$= \frac{4.32}{.0676}$$

$$= 63.89$$

63.89 64 per study group

Allowing for a 20% drop-out rate sample size was adjusted as follows:

$$n_a = 64$$

= 80 subjects per group - final sample size needed per group.

## APPENDIX 4

# Smoking Habits and Mucosal change (Survey Questionnaire)

	2	Code	No.:
			Date:
Var. No.	Variable Name	Response	
Ā.	Personal Data		
A 1	Name:		
A 2	Address:		
A3	Age:		
A 4	Marital status :		
A 5	Occupation:		
A 6	Educ. level:		
A7	No. of live children:		ari
A 8	Province of origin:		
A 9	Usual income/mo.:		
Enum	erator:		

	Code No		
Var. No.	. Variable Name Response		
B.	Tobacco usage	6	
B10	Type of habit		
B11	Reason for preference		
B12	Age started smoking		
B13	How was habit developed?		
B14	Frequency of smoking per day?		
B15	Number of sticks per day		
B16	Inhalation practice		
B17	Periods of tobacco abstinence		
B18	If B18=Y, duration(s)?		
C.	Alcohol consumption		
C19	Drinks alcohol	×	
C20	Combines alcohol with smoking		

Code No.	
----------	--

Var. No.	Variable Name Response	-
D.	Oral examination	-0
D21	Dental status:  completely dentulous  partially edentulous  completely edentulous	
D22	If edentulous, presence of dentures	
D23	Usual reason for dental consultation	
D24	Oral hygiene practice	
D25	Condition of palatal mucosa(clinical)	
D26	Exfoliative cytology results	_
D27	Photograph: Number: Batch:	

## APPENDIX 5

CODELIST FOR RESEARCH DATA: Reverse Smoking vs. Conventional Smoking

variable name	type	code
1. num	N	input code number on data sheet
2. name	C	input name of subject
3. area	N	1 = Bagong Buhay
		2 = Kalikid Norte
		3.= Kalawagan
		4 = Komunal
		5.= Batangas
		6.= Patalac
		7 = Cabanatuan proper
		8 = Camp Tinio
		9 = Cabu
4. age	N	input age of subject at time of survey
5. mstat	N	1 = currently married
		2 = single
		3 = separated
		4 = widowed
6. occup	N	1 = farmer; farmer/housewife
		2 = laundrywoman
		3 = housekeeper
(4)		4 = others(e.g. midwife,
		businesswoman)
7. educ	N	0 = no schooling
		$1 = \langle \text{level } 4 \rangle$

			2 =	> level 4, < level 6
			3 =	> level 6, < high school
8.	kids	N	input	number of children
9.	orig	N	1 =	Ilocos
			2 =	Pangasinan
	r.		3 =	Nueva Ecija
			4 =	Bulacan; Quezon province
			5 =	Batangas
			6 =	Visayas
10.	income	N	input	monthly income
11.	smkhab	N	1 =	reverse smoker
			0 =	conventional smoker
12.	filter	N	1 =	with filter
			0 =	without filter
13.	source	N	1 =	manufacture
			0 =	homemade
14.	whyhab	N	1 =	more satisfactory, no disturbance
			2 =	warmer, stronger
			3 =	milder, less hot
			4 =	afraid of heat
			5 =	no particular reason
15.	howhab	N	1 =	taught by mother/elders
			2 =	advised by birth attendant
			3 =	influenced by husband
			4 =	influenced by peers/neighbors
			5 =	just by self
			6 =	to drive away mosquitoes
16.	agesmk1	N	input	age of first puff experience
17.	yrssmk	N	input	duration of smoking in years

18. fqsmk	N	input frequency of smoking per day as
8		declared by subject
19. stksday	N	input number of sticks consumed per
		day
20. inhal	N	1 = shallow
		2 = deep
21. abstin	N	1 = with periods of tobacco
		abstinence
		0 = no period of tobacco abstinence
22. mosabs	N	input number of months abstained
23. alcin	N	1 = drinks alcoholic beverages
		0 = does not drink alcohol
24. alcsmk	N	1 = combines alcohol and smoking
		0 = does not combine
25. teeth	N	1 = completely dentulous
		2 = partially edentulous
		3 = completely edentulous
26. dentrs	N	1 = with denture
		0 = without denture
27. OHP	N	1 = toothbrush
		2 = guava twigs
		3 = areca nut husk
		4 = toothbrush and guava twigs
		5 = gurgle
28. clinpal	N	1 = with change
9		0 = without change